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ACUTE MECHANICAL OBSTRUCTION OF THE SMALL BOWEL*

Its Diagnosis and Treatment

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BOSTON

IN 1908 Scudder[1] reviewed the cases of acute mechanical obstruction of the small bowel operated on at the Massachusetts General Hospital between 1898 and 1907. Richardson[2] in 1920 and McIver[3] in 1932 reviewed comparable series of cases. This, then, is the fourth report from this hospital. Such a series of reports presupposes a continuity of active interest on the part of the visiting and house staffs in the diagnosis and management of this group of cases, and affords an unusual opportunity for the comparison of the results of treatment over a period of forty years.

Strangulation obstruction was early recognized as the greatest factor in the high mortality of these cases, and the teaching of early diagnosis and early operation has been thoroughly instilled into each and every member of the hospital staff, throughout the last thirty years. On the other hand, Scudder,[1] Jones,[4] Richardson,[2] McIver[3] and others have constantly pointed out the need of accepting the hazards of gangrene in certain cases with late obstruction, and of doing a simple drainage of a distended loop of bowel without exploration as a means of relief.

Monks[5] in 1905 suggested that a distended loop of bowel could be most satisfactorily emptied by threading it on a glass tube. Wangensteen[6] in 1931 suggested constant suction applied to an inlying stomach or duodenal tube, and reported 3 cases of acute obstruction which were relieved following this procedure. He, probably more than any other one person, is responsible for the development of the nonsurgical decompression of a distended bowel.[7–9] The difficulties of decompressing the lower segment of small bowel through a tube in the upper gastrointestinal tract were well recognized by him and others. In 1934, Miller and Abbott[10] overcame this, and they with Johnston[11] and others[12–14] have given a new impetus to the conservative treatment of these cases by combining the principle of Monks with that of Wangensteen. They have shown conclusively that a long, flexible rubber tube of special design placed in the stomach will pass through the pylorus and work its way down to the obstructing point, emptying the bowel of gas and liquid contents, the collapsed bowel threading itself on the tube as Monks had done at operation.

It is not surprising that many of us in this hospital should view with some alarm these recent enthusiastic reports in favor of a conservative form of treatment, fearing lest much be lost which has been gained by the teaching of early diagnosis and early operation. Therefore, before adopting a method so different in concept from the present teachings, it has seemed imperative to bring the earlier work of Scudder, Richardson and McIver up to date, and to study our more recent cases in an especial endeavor to evaluate, if possible, the results of their teaching, and to clarify more completely in our own minds the indications for early operation as against delayed or nonoperative treatment.

In this review we have not followed exactly the course of our predecessors. We have restricted our cases to definite acute mechanical obstruction of the small bowel occurring in patients with or without previous operation, exclusive of obstruction secondary to external hernia. We have not included obstruction of a plastic nature arising in the course of such a condition as peritonitis, as there is no question but that this type of obstruction is ideally treated by suction; nor have we included cases secondary to obvious carcinomatosis, or occurring as complications of chronic disease where the obstruction is of secondary importance. There are available for study 136 cases treated during the last fifteen years.

In addition to ascertaining the comparative re-

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sults during the last four decades, it was hoped that this study might suggest answers to the following questions: What are the factors from which an early and accurate diagnosis can be made? How important and how frequent is strangulation of the blood supply to the obstructed loop? Can one with reasonable assurance distinguish between simple and early obstruction with strangulation? Is there any distinct time after the development of obstruction when one can safely say that strangulation obstruction will not occur? What factors other than strangulation contribute to the high mortality? Are there any criteria by which one may say that immediate operation is indicated, or that conservative treatment, either as a preliminary or an active form of management, should be undertaken?

Diagnosis

History

Seventy per cent of all the patients in this series had had some previous laparotomy, and if one excludes the infants with intussusception, 80 per cent of all our patients had had a previous abdominal operation. The length of time between the operation and the development of obstruction is of no significance, in 1 case having been thirty-four years. Six per cent of our patients had been operated on for a previous obstruction, and 20 per cent had had symptoms suggestive of previous attacks with spontaneous recovery.

Symptoms

The commonest presenting symptom was pain, being the chief complaint in 98 per cent of our cases. It was usually sudden in onset (80 per cent) and colicky. Though one might well expect small-bowel pain to be at or above the umbilicus, it was described as upper abdominal in 35 per cent of the cases, lower abdominal in 35 per cent, generalized in 20 per cent and in various locations in 10 per cent. The characteristic cramplike pain may, as the obstruction progresses, become steady, and in a few cases it entirely disappeared.

Vomiting was present to some degree in 93 per cent of the cases. In many, however, it was not a prominent symptom; the so-called fecal vomiting that one is accustomed to associate with small-bowel obstruction was a late symptom.

Characteristically there was cessation of either movement of the bowel or passage of gas by rectum following the onset of pain. In 60 per cent of our cases there was nothing passed by rectum after the early symptoms of the obstruction. However, in the remainder of the cases small amounts of gas or feces were frequently passed, particularly after enemas, and in a few the bowels moved well shortly after the onset of the obstruction.

Symptoms may arise at any time in the day or night. Not a few of our patients were awakened from a sound sleep by the onset of their obstructive pain.

Physical Examination

In the early stages of obstruction there is little effect on the general condition of the patient.

As has been pointed out by Donaldson,[15] Morton,[16] Herrin and Meek[17] and others, distention comes late in the course of the disease and should not be awaited for a diagnosis. There was no discernible distention in 40 per cent of our cases.

Tenderness is not a prominent symptom, although in most cases it is present in some degree, particularly in relation to the point of obstruction. In late cases, especially those of strangulation obstruction, tenderness and spasm are usually found. This will be discussed more in detail in the section on the differential diagnosis of simple and strangulation obstruction.

Obstructive peristalsis, elicited with the stethoscope and characterized by periodic high-pitched tinkles, often though not always synchronous with colicky pain, was found in 73 per cent of our group. It must be remembered, however, that in the later stages of obstruction the abdomen may frequently be relatively silent, and long periods may elapse between peristaltic noises, which in many cases may be quite faint. Twelve per cent of our patients were described as having normal peristalsis, and it was stated to be absent in 15 per cent.

Laboratory Findings

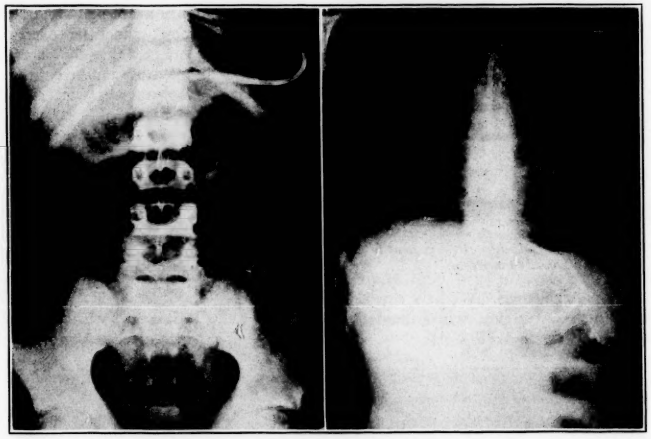
The temperature is usually normal even after the obstruction has been present for some time, unless strangulation obstruction and resulting peritonitis are present. The pulse in the earlier stages of the disease is normal or only slightly elevated. Even in the early stages there may be no change in the character or rate of the pulse. A rapid pulse in a patient with intestinal obstruction is indicative of a serious complication.

The white-blood-cell count is variable. While most commonly 15,000 or below, it varied from 3000 to 38,000 in our cases. The value of the white-cell count as a differential diagnostic sign of simple and strangulation obstruction will be discussed later.

As regards blood constituents, variations from the normal depend on the duration of obstruction, with consequent dehydration and chloride loss. In early obstruction, alterations are not expected and

are not found, but in late stages the nonprotein nitrogen rises and the chlorides fall. This rise of nonprotein nitrogen caused a mistaken diagnosis in a fatal case in this series, the patient having been judged to be in renal uremia.

The so-called "scout" x-ray film of the abdomen is the most important single objective finding in the patient with small-bowel obstruction. Its significance was first stressed in this country by Case[18] in 1915, but its use did not become prevalent until the present decade. Its value as a diagnostic sign cannot be overemphasized, particularly if the surgeon or radiologist has had experience in the interpretation of the plates. Two views are generally taken, the first with the patient supine, and the second in the sitting position. The former position (Fig. 1 *A*) is superior for the study of the gas patterns of the bowel; the latter (Fig. 1 *B*) is better for the determination of fluid levels. It is probably not saying too much to state that a close study of such films in association with a carefully taken clinical history should result in an accurate diagnosis in a very high percentage of cases. In 92 per cent of the cases in this series in which x-ray films were taken there was fairly positive evidence of obstruction. In the remaining cases the evidence was suggestive. It is our experience that x-ray films should be taken after the stomach has been emptied of gas and fluid and without the giving of an enema, because of the gas which may be injected into the large bowel and thus confuse the picture. It is not within the province of this paper to go into the details of the x-ray findings.[7, 19–23] Suffice it to say that distended coils of small bowel were demonstrated in one of our cases three hours after the onset of symptoms. On the other hand, the characteristic step-ladder arrangement (Fig. 2) is found only in the later stages. Diagnosis should be made, given the opportunity, long before this occurs, just as it should be made before the development of fluid levels. It should be stated, however, that there are times when the x-ray picture is most confusing. Small amounts of air may be found scattered through the bowel below an obstruction. In early cases a second film taken after an interval of six or eight hours may be of great help when there is doubt as to the diagnosis or as to the progress of the condition. There are other times when the shadows are such as to be difficult of interpretation. We have seen the small bowel so dilated as to be interpreted as a markedly distended cecum (Fig. 3 *A*). In certain conditions of this type the giving of a small amount of barium by rectum may entirely clarify the picture with little or no risk to the patient (Fig. 3 *B*).



A B

FIGURE 1.

Scout films in lying (A) *and sitting* (B) *positions, showing gas shadows and fluid levels in distended coils of small bowel.*

We cannot agree with Wangensteen[9] that dilatation of the small bowel is rarely seen in cases of obstruction of the large bowel. We have repeatedly seen it in such cases, even when the lesion was as low as the sigmoid (Fig. 4).

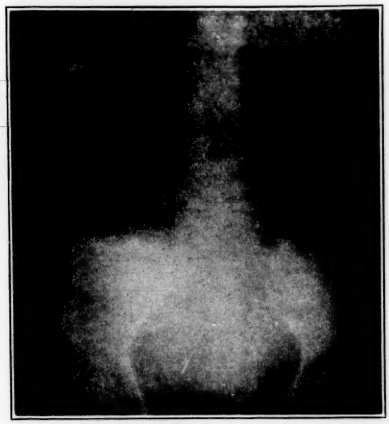


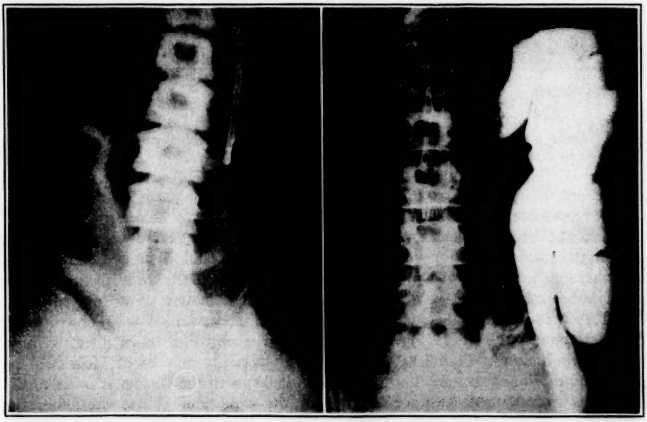
FIGURE 2. *Characteristic Step-Ladder Arrangement in Late Small-Bowel Obstruction.*

In summary, then, we may say that the five important early diagnostic criteria in acute mechanical obstruction of the small bowel are these: a history of sudden and usually severe abdominal pain, colicky in nature, particularly in a patient with previous laparotomy; vomiting, usually following the pain, which may be small in amount or in the later stages copious and fecal; evidence of one or more distended loops of small bowel, as shown by a scout x-ray film of the abdomen; and active, high-pitched, tinkling peristalsis, audible over the abdomen.

Simple Versus Strangulation Obstruction

The importance of the recognition of early strangulation obstruction cannot be overemphasized. Wangensteen[8, 9] believes that the experienced observer can accurately distinguish between obstruction which is simple in character and that with early interference with the blood supply to the obstructed loop. He places strong emphasis on the presence or development and extension of abdominal tenderness and spasm in a case where the findings are those of acute obstruction. The suggestion of a mass felt through the abdominal wall or on pelvic or rectal examination is in his judgment confirmatory evidence of a strangulation obstruction. Recognizing our own inability in the past to make this differential diagnosis, we have carefully studied these cases with this in mind.

Pain in strangulation obstruction may be more abrupt in onset and more severe, and is more apt to be quite steady with exacerbations or to change



A B

FIGURE 3.

In A, *the large gas shadow in the right abdomen was interpreted as cecum. In* B, *taken after a barium enema, the unrotated cecum is seen on the left. The distended coil on the right is small bowel.*

from colicky to steady with the onset of strangulation. In fact, in 5 of our cases the onset was so sudden and the associated tenderness and spasm so marked that a diagnosis of perforated viscus was made.

Gangrene may develop very quickly. The nature of the obstruction may be such as to strangulate the supply to the obstructed loop in the early minutes of the process. One of our patients who entered the hospital three hours after the onset of pain, and who was promptly operated on after the usual emergency ward routine of diagnosis and preparation, showed at operation a gangrenous

FIGURE 4. *Distended Loops of Small Bowel Secondary to Obstruction of the Sigmoid from Carcinoma.*

segment of bowel that required resection. Moreover, the transition from simple to acute strangulation obstruction may occur with little or no warning in a patient under observation, and this transition may happen as well between 7 p.m. and 7 a.m. as during the hours of more careful observation. It has been suggested that if forty-eight to seventy-two hours have passed with no sign of strangulation having been elicited, the chances are that there is or will be no strangulation. Half of our patients with strangulation obstruction requiring resection were operated on following a delay of six hours up to several days after an admission history of obstruction of three or more days' duration. This we feel justified in interpreting as evidence that a diagnosis of strangulation obstruction could not have been, and from the record was not, made at the time of admission, and yet resection of a strangulated loop of bowel was later necessary. We may assume, therefore, that the interval that has elapsed between the onset of symptoms and the arrival of the patient at the hospital, even though it exceeds seventy-two hours, cannot be safely taken as evidence against the subsequent development of a strangulation requiring resection.

It has been our impression that an elevation of the white-cell count above 18,000 is strongly suggestive of an interference with the blood supply to the segment of obstructed bowel. A study of the counts in these cases, however, does not confirm this. Although 35 per cent of the patients with suspected strangulation obstruction had white-cell counts of 20,000 or more, 32 per cent of this subgroup showed no evidence of strangulation at operation, and interestingly enough, the highest count in the series, 38,000, occurred in a case of simple obstruction. Even though 50 per cent of the patients with simple obstruction had white-cell counts of 15,000 or less, so many of the cases requiring resection had counts between 3000 and 15,000 that relatively little help may be expected from the leukocytic count. In spite of what has been said, we believe that a rising white-cell count should be interpreted as suggesting strangulation of the obstructed loop.

As previously suggested, a rapid pulse suggests a serious complication in a patient with intestinal obstruction. With but few exceptions, patients requiring resection had a preoperative pulse of 100 or above. On the other hand, it is of even greater significance that in the early stages of strangulation, before its toxic effects are manifest, the pulse will not be altered, and therefore the evidence of elevated pulse can be taken as evidence only of late strangulation, and is of no diagnostic value in differentiation in the earlier stages.

Tenderness, as pointed out by Wangensteen,[8] is of the greatest significance. On the other hand, as he also points out, there will be localized tenderness over the point of obstruction if this is at such a place that the examining fingers can exert direct pressure over it. Therefore, except in the hands of the most experienced surgeons we do not believe that this is a safe criterion to follow, unless one assumes that any tenderness in a patient with a diagnosis of intestinal obstruction, be it localized or spreading, is to be taken as evidence of strangulation and immediate operation advised. We can therefore only conclude from our own experience and from a thorough review of these records that we ourselves are not able, and those surgeons at the Massachusetts General Hospital under

whose care these patients have been were not able, with any high degree of accuracy, to differentiate the early stages of strangulation and simple intestinal obstruction.

Mechanism of Obstruction

It has been our impression that acute small-bowel obstruction of the type under discussion usually occurred at a single point and was most frequently caused by a band arising from previous operation or by a point of adhesion, secondary either to trauma at previous operation or to some preexisting disease (Table 1). Under single-point obstruction we include that secondary to a Meckel's diverticulum, to intussusception or to a foreign body within the lumen of bowel. A review of these cases seems to substantiate this belief, since 81 per cent of the total number of cases had some single point, the release of which was adequate to relieve the obstructing mechanism. This is of no little importance in considering the management of these cases. A single-point obstruction more completely predisposes the development of gangrene of a segment of bowel than is the case where the obstruction is more adhesive and where longer segments of the bowel are involved. Moreover, the release of such an obstruction is an extremely simple surgical procedure, and if it is done under conditions which are reasonably favorable and at a time before complications have developed, can be accomplished at very small risk to the patient.

Table 1. *Mechanism of Obstruction.*

Cause of Obstruction	Cases with Previous Operation	Cases without Previous Operation	Total
Band	48	7	55
Adhesions	17	5	22
Intussusception	1	17	18
Volvulus	8	4	12
Foreign body	2	5	7
Internal hernia	2	0	2
Not stated	16	4	20
Totals	94	42	136

Results

One hundred and thirty-six cases fulfilled the selection basis outlined above. Eight patients were never operated on because of early recovery, either spontaneously or under conservative treatment. There was no x-ray diagnosis to be found in the records of these cases, but the clinical evidence of obstruction was strong.

Twenty-seven of the 136 patients died, a hospital mortality of 20 per cent. Four of these deaths were in patients not operated on. One occurred soon after admission; the patient, already in rather poor condition, vomited a tremendous amount of foul gastric residue, and during passage of a stomach tube aspirated some of the fluid and died. The other 3 deaths that occurred without operation were from strangulation obstruction because the diagnosis was made too late or not at all. Twenty-three (18 per cent) of the 124 patients operated on had a fatal termination. The vital facts relative to the fatal cases are given in Table 2. We shall now attempt to analyze the contributing factors.

Age

The average age of our patients was thirty-three. The average age of those who recovered following operation, however, was twenty-eight, whereas that for the fatal cases was fifty-three. It is strongly suggested that obstruction in the aged should be managed differently from that in the young or middle-aged, and if one studies Table 3 one cannot but be impressed with the fact that 14 (64 per cent) of the 22 patients sixty years of age or over died, whereas only 13 (11 per cent) of the 114 patients under sixty did so. While the number of cases is too small to be conclusive, it seems significant that 75 per cent of the 8 patients seventy years of age or over died following operation. There were, of course, other factors, but when one faces a mortality of 64 per cent in any given group, one should seriously consider some change in the form of therapy used.

Table 3. *Relation Between Age and Mortality.*

Age	No. of Cases	No. of Deaths	Mortality Rate %
All patients	136	27	20
Under 60 years	114	13	11
Sixty years or over	22	14	64
Seventy years or over	8	6	75

Previous Operation

McIver,[3] among others, has remarked that the mortality of patients with mechanical obstruction who have not been previously operated on is significantly higher than that of patients who have been. This is borne out in our own series, as evidenced by a mortality of 37 per cent in those cases without previous operation, as compared to 15

Table 4. *Data on Fatal Cases in Relation to Previous Laparotomy.*

Data	Cases with Previous Operation	Cases without Previous Operation
Mortality	15%	37%
Average age	28 yr.	49 yr.
Average delay of operation in deaths	1.3 days	3.2 days
Average duration of obstruction	2.2 days	4.0 days
Delayed operation	46%	65%

TABLE 2. *Data on Fatal Cases.*

Hospital No.	Age	Previous Operation	Duration of Obstruction	Hospital Delay (More than 6 Hours)	Type of Obstructing Mechanism	Strangulation	Significant Distention	Type of Operation	Cause of Death	Postoperative Survival, or If No Operation, Post-Admission Survival
	yr.									
127478	32	Yes	5 days	None	Band	No	Yes	Lysis of adhesions	Uremia; chronic glomerulonephritis*	6 days
170387	57	Yes	1 day	None	Band	No	Yes	Lysis	Massive pulmonary collapse	2 days
279097	73	None	5 days	None	Band	No	Yes	Ileostomy	Peritonitis*	1 day
271350	85	None	7 days	None	?	?	Yes	Ileostomy	Bronchopneumonia (?); peritonitis	2 days
55785	62	Yes	9 days	4 days	?	?	Yes	Ileostomy	Peritonitis	15 days
325348	65	None	9 days	1 day	Enterolith	No	Yes	Ileostomy	Peritonitis*	1 day
251400	23	Yes	1 day	1 day	Band	Yes	No	Lysis and ileostomy	Peritonitis (?)	4 days
325062	77	No	7 days	None	Enterolith	No	Yes	Removal and ileostomy	Peritonitis; cardiac hypertrophy; early bronchopneumonia*	1 day
309543	80	No	1 day	1 day	Enterolith	No	Yes	Removal	Coma; uremia	1 day
310627	61	Yes	3 days	None	Band	Yes	Yes	Lysis and suture of necrotic intestine	Bronchopneumonia	5 days
304080	37	None	10 days	6 days	Congenital sac (?)	No	Yes	Enteroenterostomy	Peritonitis (?); pulmonary embolus (?)	2 days
334910	59	Yes	1 day	½ day	Volvulus	No	Yes	Lysis and enteroenterostomy	Peritonitis*	19 days
59287	50	Yes	14 days	1 day	Adhesions	Yes	Yes	Resection, anastomosis and ileostomy	Peritonitis*	15 days
252529	41	Yes	3 days	3 days	Volvulus	Yes	No	Resection	Peritonitis*	2 days
286189	16	No	1 day	3 days	Volvulus	Yes	No	Reduction and resection	"Shock"*	1 day
273397	60	Yes	1 day	1 day	Band	Yes	No	Resection and ileocecostomy	Bronchopneumonia; peritonitis (?)	3 days
263680	61	None	1½ days	2 days	Band	Yes	Yes	Resection, ileocecostomy and ileostomy	Peritonitis*	30 min.
161834	65	None	2 days	2 days	Volvulus	Yes	No	Resection and anastomosis	Peritonitis*	1 day
339030	72	Yes	2 days	1 day	Band	Yes	No	Resection and ileostomy	Peritonitis; bronchopneumonia*	3 days
111507	90	Yes	4 days	2 days	Band	Yes	Yes	Resection and anastomosis	Bronchopneumonia; major wound sepsis	7 days
97046	6 mo.	None	1½ days	None	Intussusception	Yes	No	Resection and anastomosis	"Shock"	1 hr.
230469	6 mo.	None	2½ days	None	Intussusception	Yes	No	Resection and anastomosis	Peritonitis (?)	1 day
307951	39	Yes	1½ days	None	Volvulus with perforation	Yes	No	Resection	Peritonitis; gas-bacillus septicemia*	1 day
36558	62	None	1 day	No operation	Band	Yes	No	None	Peritonitis from gangrenous jejunum*	1 day
278235	64	None	3 days	No operation	Volvulus	Yes	No	None	Volvulus; infarction; hemorrhage*	6 or more hr.
305438	46	Yes	4 days	No operation	Volvulus	Yes	No	None	Volvulus; gangrene of small bowel*	2 days
265061	53	Yes	2 days	No operation	Adhesions	No	Yes	None	Aspiration of vomitus*	Dead on admission

*Autopsy.

per cent in those with a previous laparotomy (Table 4).

The nature of the obstruction in these patients (Table 1) may well not be the significant factor in this increased mortality. A study of these cases (Table 4) shows that in the fatal cases not previously operated on the patients were on the average twenty-one years older than those with previous operation; furthermore, treatment had been delayed on an average of two days more after hospitalization, and the duration of obstruction had been almost twice as great. This delay in diagnosis is probably partly due to the fact that surgeons have been trained to consider that any attack of abdominal pain in the presence of an abdominal scar means acute small-bowel obstruction until proved otherwise; we therefore undoubtedly think in terms of obstruction in the patient with a previous operation, whereas the absence of any etiologic factor in the patient without previous operation renders less likely the careful consideration of acute small-bowel obstruction as the cause of symptoms. This is particularly so in the older group of patients.

Duration of Obstruction

There has been uniform agreement that the duration of obstruction plays a leading role in the mortality,[16, 24–27] and this was shown even more strikingly than we had expected in the group of patients under discussion here. Forty-three patients were operated on within twenty-four hours of the onset of obstruction, and in this group there were no deaths. There would then seem to be no question but that any patient in whose case a diagnosis of small-bowel obstruction is made or even strongly suspected within the first twenty-four hours after onset can be operated on with great safety, and can and should be operated on immediately.

It may be of significance, however, that after twenty-four hours the additional delay does not seem to carry with it a much higher mortality. This important element of time affects other factors, such as the type of operation required, and will therefore be discussed further.

Type of Operation

It is undoubtedly not so much the magnitude of the operation that is responsible for the higher mortality where more extensive operations are required, as the advanced stage of the disease and the associated complications, such as peritonitis, known to accompany it. It is therefore interesting to note that those cases operated on sufficiently early, or under conditions permitting the simple lysis of a band or adhesive point, showed an extremely low mortality (3 per cent). On the other hand, those cases in which strangulation obstruction had occurred, requiring resection of a segment of bowel, had the highest mortality (52 per cent). Again, one sees that important factor, time, and the prominent part it plays in the type of operation in cases of acute small-bowel obstruction.

TABLE 5. *Effect of Duration of Symptoms on the Type of Operation.*

Interval Between Onset and Operation	Type of Operation		
	Lysis of adhesions	Ileostomy or sidetracking operation	Resection
	%	%	%
Twenty-four hours or less	93	5	2
Twenty-five to 48 hours, inclusive	64	8	28
More than 48 hours	33	41	23

A patient operated on within the first twenty-four hours after onset of obstruction has nine chances out of ten of requiring a simple lysis of a point of adhesion (Table 5). In the second twenty-four hours his chances are only six out of ten that a simple operation will suffice, and after forty-eight hours have elapsed they are only three out of ten.

Hospital Delay

One should probably distinguish between the delay due to the patient's late arrival and that due to the inability of the surgeon to make the diagnosis, or deliberately accepted in the management of the patient. We have gone over these cases with this in mind, and have considered a delayed operation as one which was postponed six or more hours after admission (Table 6). This

TABLE 6. *Effect of Hospital Delay on Mortality.*

Interval Between Onset of Obstruction and Admission	No Delay			Delay (6 Hours or More)		
	No. of cases	No. of deaths	Mortality	No. of cases*	No. of deaths	Mortality
			%			%
Twenty-four hours or less	42	0	–	17	5	29
Twenty-five to 48 hours, inclusive	12	3	25	12	3	25
More than 48 hours	18	6	33	23	6	26
Totals	72	9		52	14	
Averages			12			27

*We have omitted 3 fatal cases of strangulation obstruction in which the patients were not operated on because when diagnosis was made after a delay it was too late. We have also omitted 8 patients who recovered without operation. This is to facilitate the comparison of operative statistics in cases of delay and no delay; as it happens, this does not affect the mortality statistics.

delay has occurred either because of failure to make the diagnosis, or because the surgeon in charge was under the impression that the patient was improving either spontaneously or with suction, and that operation was therefore unnecessary. As a

matter of fact, almost without exception any case in which there was a delay of six hours was not operated on until twenty-four hours or more had elapsed. The average hospital delay in this series was two days.

A study of these cases would seem to be essential to the formulation of proper management in the future. Forty-two patients admitted to the hospital within twenty-four hours of onset of symptoms were operated on immediately, with no deaths, whereas 5 (29 per cent) of 17 patients who entered the hospital within twenty-four hours of the onset of symptoms, but for one reason or another were not operated on as emergencies, died. However, one sees in the second twenty-four hours a mortality that is about the same with or without delay, whereas after forty-eight hours had elapsed the gross mortality of the 23 patients where operation was delayed was smaller than that in the 18 cases where immediate operation was done. It would therefore seem justifiable to conclude, regardless of other factors, that immediate operation should be done when one sees a patient less than twenty-four hours after the onset of symptoms, that early operation may be done in the second twenty-four hours, but that after forty-eight hours have elapsed immediate operation should not be undertaken.

Strangulation

There seems to be no question in anyone's mind that the most serious complication in the type of obstruction under discussion is the strangulation of the circulation to the loop of obstructed bowel. There is, however, a slight difference of opinion as to the accuracy with which early diagnosis can be made in this complication, as to how frequently it occurs and as to what part it plays in the total mortality in cases of obstruction.

We have already considered the differential diagnosis between simple and strangulation obstruction, and are forced to admit that in our hands a differential diagnosis can be made with reasonable accuracy only in the later cases. Strangulation obstruction occurred in 33 per cent of all our cases. A comparison with the cases of the previous decade, as reviewed by McIver,[3] shows that this is a distinct drop from the figure of 50 per cent in a comparable group of cases. That it is the most important factor in the fatal outcome is evidenced in our own cases by the fact that 16 of the 27 deaths were referable to gangrene. Thus 59 per cent of the fatalities were the result of interference with the blood supply to the involved loop. This is comparable to Wangensteen's[8] series (excluding hernia), where 60 per cent of the deaths classified as related to obstruction were due to gangrene.

Comparison of the Results by Decades

We believe it to be of no little importance to compare the results reported from this hospital in the three preceding decades with those in the last ten years (Fig. 5*). We see a striking drop

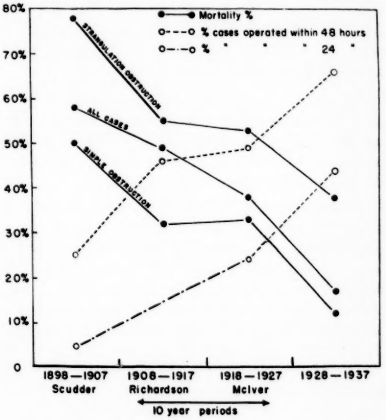


FIGURE 5.

The graph shows clearly the progressive decrease in mortality over a forty-year period. The greatest drop has been in the last decade. This decrease has come simultaneously with the increase in the proportion of cases operated on within forty-eight and, particularly, twenty-four hours of the onset of symptoms.

in mortality in the cases of both simple and strangulation obstruction. Many factors contribute to this marked and progressive improvement. However, it would seem justifiable to suggest the following:

The results of improved surgical treatment are reflected more in the cases of simple than in those of strangulation obstruction.

The increase (20 per cent in the last decade) in the number of patients operated on within twenty-four hours after the onset of symptoms is reflected in the lower mortality among the cases with strangulation obstruction.

The most productive group for further improvement is that with strangulation obstruction, and inasmuch as most of these patients die of peritonitis secondary to the pathologic process rather than to any technical surgical error, earlier diagnosis and operation before gangrene and peritonitis have ensued would seem indicated.

*Figure 5 reports the mortality of a ten-year period immediately after McIver's report. All other statistics and tables refer to a fifteen-year period (July 1, 1924, to July 1, 1939).

Any method of treatment that tends to delay operation in patients entering the hospital within twenty-four hours of the onset of symptoms may be followed by an increase rather than a decrease in mortality.

Conclusions

In beginning this study it was our hope that certain vital questions in relation to the diagnosis and management of this group of cases might be clarified. We feel warranted at this time in answering these questions as follows:

In addition to a careful history-taking and physical examination, the routine use of the scout film of the abdomen should make possible a definite diagnosis of early acute obstruction of the small bowel in a high percentage of cases.

Strangulation is the most important single factor in determining the outcome of a case of acute small-bowel obstruction. It was present in 33 per cent of the total group, and was the cause of death in more than half the fatal cases.

Regardless of Wangensteen's conviction that a differential diagnosis can be made of early strangulation and simple obstruction, we are forced to conclude that we ourselves were unable to make this differential diagnosis in early cases, and that certainly it was not made early enough to prevent resection in a substantial proportion of the cases reviewed by us.

There is no definite period beyond which one can say that strangulation obstruction will not occur. One can say that it may occur within four hours of the onset of symptoms, that operation done within the first twenty-four hours is almost certain of success regardless of the presence or absence of strangulation, and that after forty-eight hours have elapsed while the danger still exists, the advantages of delay probably outweigh the risk of strangulation.

Certain factors other than strangulation contribute to the high mortality. Though these are varied, the strongest ones are age and the duration of the obstruction. In addition the presence of distention of the small bowel at operation presents a mechanical difficulty which has been a factor in 30 per cent of the deaths in this series. One can therefore add to the triad of gangrene, age and duration, suggested by McIver,[3] the mechanical factor of distention.

The one positive essential for operation is that the patient present himself for treatment within the first twenty-four hours after the onset of symptoms. It seems doubtful from a review of these cases that any complication relative to the obstruction itself would justify delay of more than a few hours in the preparation of such a patient for operation. Almost certainly any such factors as shock, rapid pulse, tenderness and distention would be the result of strangulation obstruction, and immediate operation at the earliest possible time would be imperative. After twenty-four hours have elapsed, other factors such as distention, dehydration and chemical imbalance rapidly assume importance equal to or surpassing the risk of strangulation. Therefore, in cases where the obstruction is of twenty-four hours' duration or longer, delayed treatment would seem indicated in any patient sixty years of age or older unless there is definite evidence of strangulation obstruction.

Recommendations for Treatment

There is probably no condition in which the results to be obtained depend so much on experience, judgment and individual treatment as that in cases of acute mechanical obstruction of the small bowel. However, in view of the above findings, and judging from the experiences of Wangensteen,[6–9, 19] Miller, Abbott and Johnston[10, 14] and others, as well as from our own, we feel justified in suggesting the following principles for the management of such cases:

The degree of dehydration and chemical imbalance should be evaluated and appropriate methods instituted for relief, with observance of the necessity of transfusions for certain patients with strangulation obstruction.[28, 29]

A double-lumen Miller-Abbott or similar tube should be passed immediately, to be left down whether or not operation is undertaken.

Immediate operation is indicated in any case examined within twenty-four hours after the onset of symptoms. We see no place for conservative treatment in this group.

Early operation should be done on a patient seen in the second twenty-four hours after onset.

Delayed or nonoperative treatment is indicated in the absence of definite signs of strangulation obstruction in any patient seen later than forty-eight hours after the onset of symptoms, or in any patient over sixty years of age, unless the obstruction is of less than twenty-four hours' duration and the patient is in good condition, without distention.

If conservative treatment is instituted, the following routine should be followed: serial abdominal films at twelve-hour to twenty-four-hour intervals, in order to follow the progress of the tube and the degree of distention; bi-daily white-cell counts*; and frequent abdominal ex-

*Even though normal or low white-cell counts do not exclude strangulation, we think that a rising count should be accepted as evidence of increasing interference with the blood supply of the involved segment of bowel.

amination, at least every four or six hours, with the examiner particularly alert for the development of point or rebound tenderness.

If decompression cannot be accomplished by intubation, and laparotomy under general or spinal anesthesia seems too hazardous, the distended bowel should be drained by inserting a catheter in a distended loop under novocain anesthesia. No attempt at exploration should be made at this time.

If decompression is successfully accomplished and normal bowel action re-established, it is our belief that for a good-risk patient an operation of election at the same hospital admission should be given serious consideration. We believe that since the obstructing mechanism is probably still present, and since 26 per cent of our patients have given a history of previous attacks, the risk of lysis of the point of obstruction is less than that of future obstruction, unless the patient is assured of competent surgical care within the first twenty-four hours of any subsequent attack.

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Discussion

Dr. Richard Thompson, Boston: I brought with me one of the Miller-Abbott tubes, and will speak briefly of how we use it. It has a very thin balloon at the end. The tip beyond this balloon is metal and perforated, and is connected with one of the lumens that run throughout the whole ten feet. The other lumen goes to the inside of the balloon. The balloon can be inflated and held inflated by the clamping off of the other limb at the proximal end. We lubricate the balloon with surgical lubricant because oil or grease will injure the rubber, and pass it through the nose to get it into the stomach. The technic of getting it beyond the pylorus is sometimes quite troublesome, and we have had some experiences which have helped us to learn how it may be worked through.

Of course x-ray examination, by using the fluoroscope, or a flat plate if the patient is too ill to be moved to the x-ray department, is of great value. If one is in extreme haste to get the tube down, fluoroscopy is very important; however, if for any reason one wishes to use the tube for purely diagnostic purposes, or does not feel anxious to get it down immediately because the obstruction is subacute, the patient may simply lie in bed in any position, not necessarily on the right side, and take cold, preferably ice-cold, fluids by mouth. If the tube is given a little urging through the nose, it will usually pass the pylorus.

Of course, as Dr. McKittrick said, the ideal place for the use of this tube is in early postoperative obstruction, when the condition is due to plastic exudate about the bowel, and when one feels perfectly sure that there is no constricting band that may cause gangrene of a loop. After the tube has been passed in such cases, recovery is made easier, because we can give fluids, and even milk and soft solids, at a much earlier time than we should dare to without the safety-valve effect of the tube in the small bowel.

Certain patients who obviously have something wrong in the small intestine, perhaps regional enteritis, can be very effectively diagnosed with the Miller-Abbott tube.

Dr. S. Peter Sarris, Boston: There is one aspect of the treatment of acute mechanical obstruction of the small bowel that Dr. McKittrick did not mention: that is the use of 95 per cent oxygen in relieving distention. This method, which was introduced by Dr. Jacob Fine, of Boston, is based on sound physiological principles. The first is the observation by McIver, that the chief component of gas in the distended small bowel is nitrogen. The second principle is that gases diffuse through a semipermeable

membrane in proportion to their partial pressure. Dr. Fine therefore administered 95 per cent oxygen, which washes the nitrogen first out of the lungs and then out of the blood stream. Finally there begins a diffusion of nitrogen from the intestines into the blood stream. As a result, distention is diminished within twenty-four hours in about 60 per cent of such cases.

We have not used this method extensively, but have seen encouraging results from it. We suggest that its outstanding value lies in supplementing the treatments outlined by Dr. McKittrick. Whether one is going to operate or to use the Miller-Abbott tube, no harm will come from putting the patient in a 95 per cent oxygen tent, if he is badly distended.

I wish to say a word about the difficulties we have had with the Miller-Abbott tube. We do not profess to be experts, but there is one thing that we have learned, and which the originators of the tube themselves admit. That is that in about 15 per cent of cases it is impossible to get it through the pylorus. Another thing we have discovered is that these tubes need extremely careful attention if they are to function properly.

A complication in small-bowel obstruction is knotting of the tube. This is probably extremely rare, but it certainly occurred in one case. The tube was passed, but shortly thereafter drainage ceased. Accordingly the tube was pulled out, and as the end of it reached the pharynx the patient choked and almost died of asphyxia. Quick thinking on the part of the assistant who was drawing out the tube led him to puncture the balloon, and so allow the tube to be extricated. Apparently the tube had become knotted, and this prevented not only aspiration of the small-bowel contents but also deflation of the balloon.

Dr. Irving J. Walker, Boston: I should like to mention two rather interesting experiences with the Miller-Abbott tube. The first was a knotting of the tube in the stomach. After the tube was passed the patient was x-rayed to determine its position. One film showed what was apparently a knotting. This did not worry us much; the tube was withdrawn into the mouth, the knot was untied and the tube was passed into the stomach again.

The second incident was rather distressing. As you know, the Miller-Abbott tube is a two-way affair, one tube leading to the balloon, and the other projecting through and beyond the balloon. The connecting tips of the metal yoke do not indicate to which tube each leads. It seems to me that the manufacturer could indicate the tip leading to the tube that is to be used for inflating the balloon, and thus aid in avoiding embarrassing situations like the one that occurred in the following case. The patient was thought to have small-bowel obstruction, due to bands following a previous appendectomy. A flat plate of the abdomen showed no fluid levels. We decided to pass the tube, and did so. Eighteen hours later the balloon was well down and the small bowel was decompressed. We later operated and found an obstructive band in the ileum, which we divided. The tube was left in place for further decompression. After this had been accomplished, we resolved to feed the patient through the tube projecting beyond the balloon. Apparently the nurse tried to use the tube leading to the balloon. She soon realized that the food was not entering the bowel, and therefore shifted to the other tube. The feeding was carried out for two days, at the end of which time it was decided to remove the tube. This I undertook to do. The tube seemed to come out rather harder than usual. However, I gradually worked it up farther and farther. I then became suspicious that the balloon was not deflated and was in the stomach. I then tried to pass fluids through the tube leading to the balloon, but was unsuccessful. Another attempt at withdrawal was made, but again resistance was encountered at the cardiac end of the stomach. After considerable pulling the balloon exploded within the stomach and the tube was easily withdrawn. Later it was discovered that the tube leading to the balloon had become plugged with the feeding material. The incident worried me considerably, especially at the time traction was being placed on the balloon when it had reached the cardiac end of the stomach, because of the possibility of rupturing the lower end of the esophagus.

We have found the Miller-Abbott tube of special value in the differential diagnosis of paralytic ileus and mechanical obstruction. I refer to those patients who four or five days after operation show distention, and more particularly to cases in which there is infection within the peritoneal cavity. Such cases often offer a problem as to whether one is dealing with a peritoneal exudate that has resulted in a kinking of the bowel, paralytic ileus or a combination of both. We have found that the tube decompresses the small bowel regardless of whether the condition is paralytic ileus or mechanical obstruction. When the bowel has been decompressed, we have passed 5 or 10 cc. of a weak barium solution through the tube that projects beyond the balloon. This at once answers the question whether one is dealing with paralytic ileus or mechanical obstruction. In the former, the barium is found by x-ray to be in the large bowel. In the latter it will not have passed beyond the ileocecal valve.

THE PREVENTION OF DIABETES MELLITUS

A Clinical Lecture

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FOR several years, as an annual event in his course on preventive medicine at the Boston University School of Medicine, my friend Dr. David L. Belding has asked me to give a lecture on the prevention of diabetes mellitus. This year I propose to thank Dr. Belding for the compliment by retaliating in print; for in order to give the kind of lecture which he would like, vastly more knowledge is needed than is at present available, and therefore I am asking my New England colleagues for assistance.

When all is said and done, peculiarly little is known of the cause of diabetes. Certainly to find an assured organic basis for the disorder is by no means easy, in spite of the excellent histological observations on the islands of Langerhans made by Opie[1] and by Weichselbaum and Stangl[2] in 1900 and 1901, and in spite of the demonstration of the hydropic degeneration of the "beta" cells of these islands in experimental diabetes which Allen[3] and Homans,[4] working independently, were able to accomplish a few years later.

Until recently, the physiological basis for believing that the pancreas has a good deal to do with diabetes seemed clearer, for in 1889, as everyone remembers, Minkowski,[5] with the aid of von Mering, showed that ablation of the pancreas in animals induces a true diabetes mellitus, with glycosuria and hyperglycemia. This discovery, in fact, eventually led Banting and Best[6] to find insulin, an extract from the islet cells of the pancreas that has made of diabetes an easily treatable condition.

The work of recent investigators like Houssay[7] and Young[8] now throws a reasonable doubt on the belief that the pancreas primarily needs to be at fault in diabetes. Last winter at the Harvard Medical School, as many medical students recall, Dr. Young showed slides from a diabetic animal which had developed a fatal diabetes although the pancreas had been left untouched. The disease was induced by injections of extract from the anterior lobe of the pituitary gland, and the resultant changes that took place in the pancreas secondary to such treatment were indeed remarkable.

Thus in diabetes we have a disease, with an underlying cause still to be identified, that produces a clear-cut, unmistakable clinical picture. Diabetes clinically, however, is such a respectable, well-studied affair that of course much is known of its natural life history, and hints have been given as to its preventability. In the sixth edition of Joslin's[9] book on the treatment of diabetes mellitus, there is an interesting chapter dealing with this phase of the disease.

Joslin comes out flat-footed: he says that heredity is the basis of diabetes. In the same volume, White and Pincus elaborate on his views. They say that the evidence that the potentiality of developing diabetes is inherited rests primarily on four factors: the almost simultaneous occurrence of diabetes in both members of pairs of similar twins; its greater incidence in the blood relatives of diabetic patients than in those of a control population; the demonstration that Mendelian ratios of the recessive type are found in a large series of cases selected at random; and the demonstration of expected ratios of a diabetic tendency in presumably latent cases.

Their figures on twins are striking. In 19 pairs of dissimilar twins, one of whom became diabetic, the incidence of diabetes in both twins was 10 per cent; in 16 pairs of similar twins, one of whom became diabetic, it was 69 per cent. These figures are all the more remarkable because in the dissimilar twin group 10 per cent of the parents were diabetic, compared with only 6 per cent of the parents in the similar twin group.

The incidence of diabetes in the parents and siblings of diabetic patients differs significantly from that in the parents and siblings of non-diabetic subjects. White and Pincus found that among 4434 parents and siblings of diabetic patients, 6.7 per cent were afflicted with the disease; whereas among 1219 parents and siblings of a group of patients without diabetes, only 1.2 per cent had it. These figures also seem convincing.

White and Pincus are as conservative as possible. They say that although the excess of diabetes in the blood relatives of diabetic patients suggests inheritance, it does not prove it, for to do so a pattern must be demonstrable. They have drawn some interesting speculation from Joslin's material, to the effect that diabetes does, in fact, follow a definite pattern of inheritance,

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and that this pattern, according to the Mendelian hypothesis, is one of simple recessiveness. On the other hand, Perkins[10] regards the inherited tendency to develop diabetes as a dominant characteristic.

Admitting all this to be true,—that the cause of diabetes is uncertain, that heredity is the basis of it and that laws of heredity govern the transmission of the disease according to the Mendelian theory and follow the pattern of simple recessiveness or of dominance,—we are not much farther ahead when we begin to speak of prevention. For we do not know what determines the onset of diabetes.

Some secondary factor seems needed to activate the diabetic gene. White and Pincus chose the anterior pituitary lobe. They believe that something happens to this mysterious little gland which makes it hyperactive. Given an individual who inherits diabetic susceptibility and let his anterior pituitary lobe for some unknown reason become overactive, and he will develop what we clinicians call diabetes.

Joslin, on the other hand, blames obesity for being the flint that when properly struck makes the spark that fires the gene that causes diabetes. And he has a great deal with which to support this theory: statistics on the frequent association between diabetes and obesity; statistics from insurance records showing that the subsequent development among persons accepted for insurance after medical examination and known not to be diabetic is far more frequent among overweights than among persons of average weight or less; statistics regarding conjugal diabetes where, in his experience, both husband and wife have been fat, the implication being that they contracted the disease from exposure to good food rather than to one another; statistics regarding the occupational incidence of diabetes—"whenever and wherever conditions of life are easy, food abundant and relatively cheap over long periods, and when large numbers of individuals become accustomed to partake of food in excess of their requirements for the expenditure of energy, the frequent development of overweight and of diabetes is favored." In other words, those of sedentary occupation and large appetite are most likely to develop diabetes; those who labor the hardest and keep lean get it the least.

There are other influences than obesity that have to do with causing diabetes. Of these, racial peculiarities are the most outstanding. The high incidence of diabetes in Jewish people is proverbial. It has low incidence among the Chinese, Japanese and Negroes, and it appears to have rising incidence among the Irish. Factors like trauma, infection, worry and arteriosclerosis do not appear important. This is about all that is known about diabetes and its etiology. The disease is a peculiar one, transmissible by inheritance, somewhat racial in character, afflicting fat people rather than thin, brought into being by an unknown factor, and perhaps more directly due to abnormal pituitary function than to abnormal pancreatic function, though pancreatic extract is the most useful therapeutic weapon at present available.

Can rational preventive measures be designed to operate against a disease about which, relatively speaking, so little is known? Joslin is optimistic. He is hopeful that the disease, to some extent at least, may be prevented by two barriers. If White and Pincus are right, a child cannot inherit a diabetic tendency from one diabetic parent alone but only from two diabetic parents, from one diabetic parent and a hereditary carrier, or from two hereditary carriers. The corollary is that to prevent diabetes, diabetic patients must not intermarry, or marry into families where diabetic carriers are known to exist; and they must remember that their children, too, must not marry diabetic persons or into diabetic families. As for obesity, Joslin says that it should be avoided generally, and particularly by the relatives of diabetic patients. Focus on the diabetic family, he says, for the prevention of the disease, remembering that all relatives, and especially the obese relatives, are the most susceptible. Hence, focus on the diabetic family to forestall intermarriage of diabetic carriers and to prevent obesity in all its members. Instruct each member not to marry into a diabetic family and never to allow his or her weight to go above the normal standard; in fact, after the middle of the fourth decade urge them to keep their weights 5 to 10 per cent below normal. If you can accomplish these things, you are doing all that can be done, in the light of present knowledge, to prevent diabetes.

I confess that I am a good deal less hopeful than is Joslin about the rationality of preventing diabetes. I present two cases to show why.

The first case is that of Mr. P. He is 56 years old, and a Yankee. He believes that his father died of "shock and diabetes," and he recalls having been told that a maternal uncle also had diabetes. Therefore, Mr. P. very obviously owns his diabetic genes by acquiring them in a most direct manner. To preclude his ever-developing diabetes, he should have been one of Peter Pan's "lost boys" and should never have been born; this would have been a hardship to him and his friends, for he has enjoyed life, been popular and accomplished much. Or possibly his parents should not have married each other, harboring as they did the power to transmit diabetes. The difficulty here is that Mr. P.'s father did not develop diabetes until he was an old man, nor did Mr. P.'s uncle. Mr. P.'s parents married when they were young, without estab-

lished diabetic family histories, knowing nothing of the disease and probably caring less, and they lived happily together for a great many years. They brought contentment and sunshine into other people's lives, for they were generous citizens. It would have been a loss to many people and even to all New England if, in order to prevent a theoretically possible son from developing a theoretically possible diabetes, they had not married.

Mr. P. inherited another interesting tendency. Both Mr. P.'s parents were stout people, and Mr. P. himself seemed doomed to a stout build. I have known him for nearly 50 years and can testify that during this last half-century he grew from a stout little boy into a stout, thick-set, muscular young man, and has mellowed into a stout, thickset older man. For the last 40 years he has been actively interested in all forms of physical exercise. He has played games regularly and violently. He has never been a gross eater, and has watched his weight with care. In fact, since 1908 he has kept a systematic record of his weight, not wishing to become too stout. During these 30 years his weight has fluctuated between a low point of 205 and a high point of 235. He has found that when he weighs too little he does not feel well, and that to suit

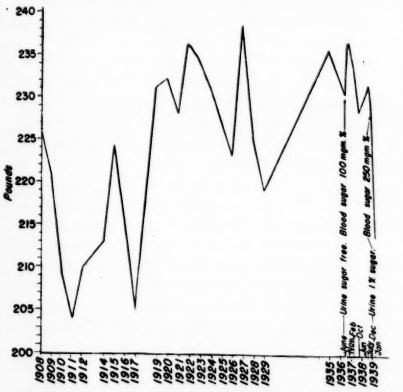

FIGURE 1. *The Weight Curve of Mr. P. during the Period of Time He Was "Prediabetic."*

his own particular sense of well-being, he can do most, with least fatigue, at a weight level of around 230 pounds. Between 1908 and 1936 he had scarcely a day's illness. Besides being active in athletics, he also has been in a position of considerable business responsibility, and to all intents and purposes has been well.

Mr. P. consulted me professionally in June, 1936. At that time he did not have diabetes; the urine was sugar-free, and after an ordinary meal the blood sugar concentration was 100 mg. per 100 cc. He came to see me again on November 29, 1938. In the interval between the examinations he had gained 10 pounds in weight and had been feeling perfectly well, leading his usually active life and having no physical complaints whatsoever. He told me that in August, 1938, he first became conscious of the fact that he was getting up two or three times at night to urinate. He denied any excessive thirst. He said that his appetite was as usual and no greater than it ever had been. The urine contained 1 per cent of sugar and the blood-sugar concentration was 250 mg. per 100 cc.; in other words, Mr. P. had developed diabetes under my eyes.

Could the diabetes have been prevented? As has been said, obviously not allowing Mr. P. to be born would have been one method, not altogether satisfactory for many reasons. Could his diabetes have been prevented by not allowing him to weigh so much? There is no way to answer this question with assurance. On the one hand are Joslin's statistics, which suggest that for Mr. P. to have avoided obesity would have been wise. On the other hand is Mr. P.'s own experience, suggesting that had he kept his weight down he would have not felt well or have been so active as he proved to be, and thus would not have accomplished so much in life or have been so happy; and it is a dangerous doctor who is willing deliberately to gamble with the happiness and effectiveness of his patients' lives unless he knows exactly what he is doing.

Finally, there is evidence for thinking that one's type of body build is largely an inherited characteristic. Mr. P. with diabetic genes also inherited obesity genes. Thus, as he grew up, had he kept his weight down by undereating he still would have been a stout, thickset individual, to be sure an artificially thin one, but nevertheless by constitution the same stout, thickset individual he was designed to be and, so far as anyone knows, with the same inherited health liabilities. It seems to me that there is no certainty, with such inherited tendencies toward diabetes and toward body build, that Mr. P. could have avoided the one by attempting to manipulate the other. In other words, I doubt whether his diabetes was preventable.

I cannot help raising one more question. Assuming that Mr. P.'s diabetes could have been prevented, would to prevent it necessarily have been wise? He had fifty-five years of active, happy living as a prediabetic before the disease eventually became apparent. He took no particular care of his health during these years, kept largely out of medical hands and took life as it came. You will remember that a great many years ago Osler[11] wrote a most delightful paper, "On the Advantages of a Trace of Albumin and a Few Tube Casts in the Urine of Certain Men above Fifty Years of Age." I am sure that Joslin could write an equally appealing paper stressing the advantages, to certain persons above that age line of a little glycosuria, for once patients of this age are conscious of the symptoms of diabetes, they seek medical advice and begin to take care of themselves. Mr. P. is a case in point. Very simple dietetic restriction after the diabetes made itself known, em-

phasis on the importance of reasonable care and insistence on occasional check-ups have resulted in the disappearance of diabetic symptoms, slight loss in weight and an increased sense of well-being. The discovery of diabetes in Mr. P.'s case will, I hope, prove to be a form of life insurance for him, as a result of which he will live longer and more happily than he could have done without it! You

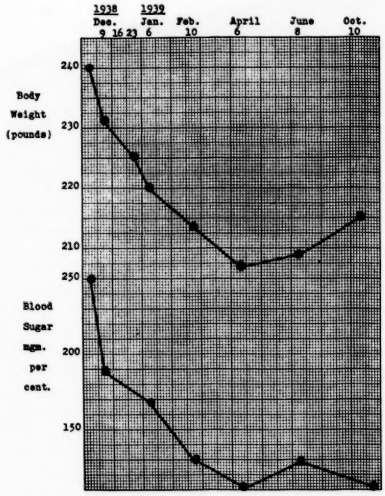

FIGURE 2. *The Advantage of a Little Sugar in the Urine of a Man above Fifty Years of Age.*

Moderate diet restriction results in weight loss, an increased sense of well-being and a disappearance of hyperglycemia and glycosuria. Diabetes may be a good form of life insurance.

can see that I am by no means convinced, even had it been possible, that to prevent diabetes here necessarily would have been judicious.

The second case is rather different. Mrs. R. is a Jewish lady, 59 years old, whom I first saw 4 years ago. She consulted me for ill-defined aches and pains. A sister had died of diabetes, so that Mrs. R. presumably was to be regarded as a potential diabetic carrier. She was obese, too, for she weighed 162 pounds, which, for her height and age, made her nearly 30 pounds overweight.

The first specimen of urine which was examined contained 2.5 per cent of sugar. However, a fasting blood-sugar sample had a concentration of 140 mg. per 100 cc.; a fasting urine sample was sugar-free. I was uncertain whether she had diabetes, but on general principles advised her to follow a restricted carbohydrate diet. I saw her 5 months later; she had lost 7 pounds, and had a blood-sugar concentration of 110 mg. per 100 cc., and an entirely sugar-free urine.

I lost track of her for a year. Then, however, she returned with symptoms suggesting gallstones. She weighed 150 pounds and looked very well. Four samples of urine were examined; two were sugar-free, and two showed the faintest trace of sugar. Stupidly enough, my interest was so focused on the gall bladder, which proved to be normal, that I neglected to determine the blood-sugar level.

A few days ago, more than 3 years after the last note, I again saw her. She weighed 154 pounds, thus revealing no great change in weight; she had no diabetic symptoms but called on me more for old times' sake than for any other reason. The urine had 5 per cent of sugar and the blood-sugar concentration was 290 mg. per 100 cc.

Could her diabetes have been prevented? At present we have the scantiest information regarding how the attack of what we recognize as diabetes commences. Both Mr. P. and Mrs. R. are interesting on this score. Mr. P. apparently developed clinical evidence of diabetes with relative acuteness, whereas Mrs. R. developed it so insidiously as to make it impossible to demarcate the nondiabetic part of her life from the diabetic.

I am in hopes that knowledge can be accumulated so that it may be possible to know more than is now known about the factors which make diabetes apparent. I am in hopes that more authenticated cases can be reported like the dramatic one of Wallach's,[12] which he described in 1866. There was a young chemist, it appears, who was in the habit of testing his own urine at least once a week, using it as a control against pathologic liquids whose sugar content was under analysis. His urine always was sugar-free. He had an attack of bronchitis, and as he was convalescing he began very suddenly to pass large quantities of urine, drink large quantities of water and lose weight. Wallach examined him and suspected diabetes, but, he wrote: "Because the full significance of this illness was so well known to my patient, tact forbade me to test his urine immediately." A few days later the young chemist made his own diagnosis. Wallach goes on: "I found my patient out of bed, sitting at his worktable, holding in his trembling hand a test tube with his own urine in it and with an ominous precipitate at its bottom." The poor chap died in coma within a few weeks of the first onset of symptoms. Here was a case beginning acutely. I, too, have seen a patient thought not to have diabetes when first she was studied who subsequently developed it with such apparent suddenness that she could remember almost the day and hour when symptoms began.

I believe that the question of preventing diabetes cannot be discussed any more logically or with any more conviction than I have discussed it here until more is known of the fundamental nature of the disease. It is to investigate this that I want help from my colleagues. Therefore,

I take this opportunity of begging them to send me case reports through which to study the possible factor or factors that induce the clinical manifestations of diabetes: large numbers of case reports on patients well studied when they were nondiabetic, with accurate accounts of the manner in which diabetic symptoms seemed first to become apparent. A large group of cases analyzed from this approach not only might enable one to classify diabetes more accurately than can be done at present, but also might perhaps bring to light some common factor running through the group which would appear to define the agent that explodes the disease in the predisposed individual. Could the beginning of diabetes be recognized and the trigger that fires it be identified, better means for prevention might be developed than those advocated by Joslin. At present, in most cases, lack of knowledge makes the assured preventability of diabetes very elusive.

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ENGORGEMENT OF THE PULMONARY VEINS BY EXTENSION OF CARDIAC ENLARGEMENT POSTERIORLY*

Relation to Postural Dyspnea in Cardiac Patients

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ENGORGEMENT of the pulmonary vessels in cardiac patients is usually attributed to failure of the left ventricle. The latter causes an increase in pressure in the left auricle, and this pressure in turn is exerted backward on the pulmonary circulation. This theory of backward pressure, however, has not appeared to be fully satisfactory. Attention tends to be too exclusively centered on the heart, and adequate consideration has not been given to the mechanical relations of the heart to the structures behind it. It is the purpose of this article to present reasons in support of the conception that compression of the pulmonary veins and of the lungs by extension of cardiac enlargement posteriorly is a more valid explanation of engorgement of these vessels and of dyspnea of cardiac origin.

ENGORGEMENT OF THE PULMONARY VEINS

Overfilling of the pulmonary veins has become practically synonymous with failure of the left ventricle, as may be documented by a citation from a recent book.[1] Thus:

> The clinical picture of isolated insufficiency of the left side of the heart is characterized by signs and symptoms resulting from engorgement of the pulmonary circuit in the absence of systemic venous engorgement and its consequence, such as edema and swelling of the liver.

The mechanism by which the engorgement of the pulmonary veins is produced is usually explained by what is known as the "back-pressure theory." Apparently this theory was developed approximately a century ago.

Corvisart,[2] one of Napoleon's physicians, in 1812 stated:

> It is possible that, in diseases of the heart, the difficulty of breathing proceeds entirely from the mechanical compression of the lungs, by the enlargement of the heart, or the evolution of an aneurysmal tumor; this is true in some cases, but in a greater number, the difficulty of respiration appears to belong solely to the accumulation of the blood in the vascular system of the lungs, from the embarrassment which it suffers on returning into the cavities of the heart, deranged wholly or partly in their natural organization.

Hope,[3] an English physician, in his book published in 1842, further developed this conception, and to

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him is generally credited the origin of the back-pressure theory. He wrote:

> But when the distending pressure of the blood preponderates over the power of the ventricle, its contents, from not being duly expelled, constitute an obstacle to the transmission of the auricular blood. Hence the auricle becomes over-distended, and the obstruction may be propagated backward through the lungs to the right side of the heart, and there occasion the same series of phenomena.

Harrison[4] in his recent book supports the back-pressure theory.

However, a considerable number of writers, among whom may be mentioned Mackenzie,[5] Lewis,[6] Starling[7] and Wiggers,[8] do not accept this explanation, save in cases of marked stenosis or insufficiency of the mitral valve. It should be noted that Hope's description contains the word "propagated"; and a reasonable interpretation of this is that the pressure is transmitted backward through the blood in the pulmonary veins. Corvisart's words, as I understand them, are limited to emphasizing the importance of cardiac enlargement and engorgement of the vascular system of the lungs, "from the embarrassment which it suffers on returning into the cavities of the heart. . . ." The quotation does not elucidate the exact mechanism of the embarrassment.

I have found it difficult to be satisfied with the applicability of left ventricular failure to attacks of acute edema of the lungs experienced by patients with chronic arterial hypertension, when prior to the attack they have been in bed and asleep, whereas in the daytime they may have been ambulatory and relatively free of symptoms. Why do such left ventricles fail, even though they be abnormal, when the patient is in bed? Rest in bed is usually believed to be the best treatment for an individual with cardiac insufficiency. Since left ventricular failure is diagnosed in life by its effects, namely by engorgement of the pulmonary vessels, one tends to consider what there may be in the horizontal posture that might cause overfilling of the pulmonary vessels.

When the patient lies down, the lessening of the operation of the force of gravity causes a better return of the blood from the inferior vena cava to the right heart. This blood is promptly forwarded to the lungs by the contraction of the right ventricle; the Bainbridge reflex may be in operation in this situation. Thus, there is reason to believe that there is a generous inflow into the pulmonary vessels when the patient is in a horizontal position. Furthermore, when he lies down the heart naturally gravitates backward and rests more on the structures behind it (below it, in the horizontal position); if the heart be enlarged, it might conceivably exert a more than normal pressure on its bed. It therefore becomes pertinent to consider the anatomic relations and physiology of the retrocardiac structures.

The Retrocardiac Space

The retrocardiac space anatomically comprises part of the middle and posterior mediastinal spaces.

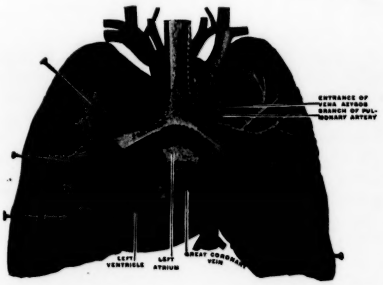


FIGURE 1. *Pulmonary Veins Seen in a Dorsal View of the Heart and Lungs.*

The lungs have been pulled away from the median line, and a part of the right lung has been cut away to display the air ducts and blood vessels. (Reproduced from Anatomy: Descriptive and applied. *Henry Gray. From the new American edition. [Philadelphia: Lea and Febiger, 1913.] By courtesy of the publishers.)*

In brief, it is the area between the posterior surface of the heart and the front of the vertebral column.

The pulmonary veins pass through it, as they return the oxygenated blood from the lungs to the left auricle of the heart. Each venous trunk is formed by the union of the lesser radicles in the lungs and is about 1.5 cm. in length; both are approximately of the same caliber. The right pulmonary veins pass behind the right auricle and ascending aorta and superior vena cava; those on the left pass in front of the thoracic aorta. These veins are thin-walled, and contain no valves.

The inner or mediastinal surface of the lungs presents a deep cavity that accommodates the pericardial sac. Above and behind this concavity is a triangular depression termed the hilus, where the structures forming the root of the lung enter and leave it.

Respiratory Movements of the Heart and Lungs

Shipley[9] states that the heart is attached to both lungs by means of the pulmonary artery and veins. It is also attached to the diaphragm at its central tendon by the pericardial covering of the inferior vena cava as it pierces the diaphragm. The heart is firmly fixed to the aorta, which is said to move

very little. Shipley points out that both the lungs and diaphragm are movable structures, and fortunately for the heart, they move in unison.

It is generally known that the heart moves with the diaphragm; its changes in position and contour with respiration are well visualized on the roentgen plate. Less attention appears to have

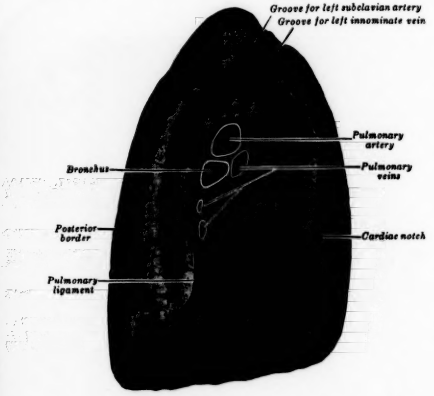


FIGURE 2. *Mediastinal Surface of Left Lung. (Reproduced from* Anatomy: Descriptive and applied. *Henry Gray. From the new American edition. [Philadelphia: Lea and Febiger, 1913.] By courtesy of the publishers.)*

been paid to the movement of the heart in relation to the retrocardiac space. Fluoroscopic examination of the effect of respiration on the proximity of the posterior border of the heart to the spine has proved a little confusing because of the multiplicity of movements — ribs, heart and diaphragm. Roentgen plates, however, clearly disclose that during inspiration the heart moves away from the spine and during expiration toward it (Fig. 4). This observation has been confirmed by fluoroscopic examination of some hundreds of patients.

Sir Arthur Keith[10] has written a detailed description of the movements associated with respiration. He finds that, contrary to the usual belief, the roots of the lungs are not fixed but move with respiration. This movement is depicted in Figure 5. Thus it can be seen that heart, lungs and diaphragm move in unison with respiration, and that the retrocardiac space is diminished during expiration.

THE FLOW THROUGH THE PULMONARY VEINS

Every drop of blood sent out by the left ventricle must pass through the retrocardiac space by way of the pulmonary veins. The flow per minute under resting conditions is given as 5 to 8 liters, and after strenuous exertion it may increase to 30 to 40 liters.[11] It is obvious that the amount of blood that must pass through the pulmonary veins varies markedly according to physiologic need.

There is a variation in the statements regarding the mechanism that causes the flow of blood through the pulmonary veins. It is believed to be aspirated by a suction action exerted by the left auricle, or to be propelled by the pressure exerted by the muscular movements brought into action by the respiratory act. Arguments have been presented elsewhere[12] which cast doubt on the validity of an aspiration or suction mechanism. Effective suction is contrary to physical laws when the

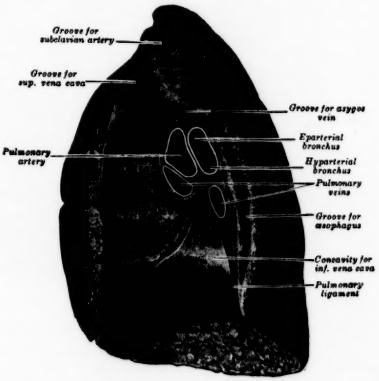


FIGURE 3. *Mediastinal Surface of Right Lung. (Reproduced from* Anatomy: Descriptive and applied. *Henry Gray. From the new American edition. [Philadelphia: Lea and Febiger, 1913.] By courtesy of the publishers.)*

aspiration must be operated through thin-walled and collapsible tubes, and the pulmonary veins are such structures. These veins bleed when severed during operations on the lungs. The problem appears to have been solved recently; the pressures existing in the pulmonary vessels have been measured in the closed chests of dogs and found to be positive. The pressure in the pulmonary veins, taken during quiet breathing, was found[13] to average 3 to 12 mm. of mercury.

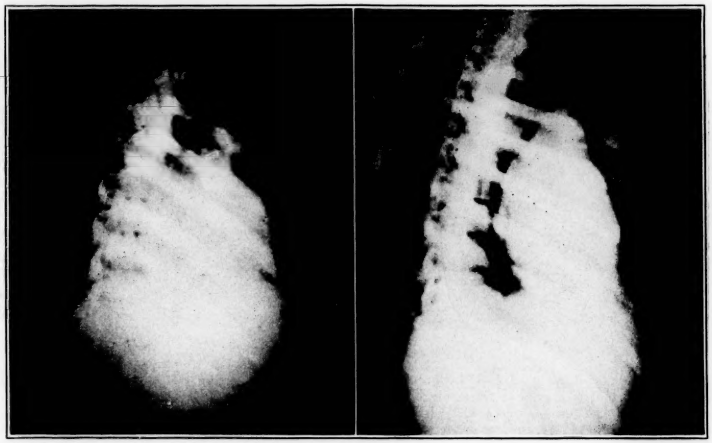
The source of this positive pressure in the pulmonary veins is of interest, particularly since it is stated that the pressure in the pulmonary capillaries is at or about zero, and that the pulmonary venous pressure changes but little in response to a rise in pressure in the pulmonary artery.[14]

The Postmediastinal Syndrome

There is a condition, sometimes termed the postmediastinal syndrome, in which dyspnea is an outstanding symptom. Tumors invading the posterior mediastinum are the usual finding at necropsy. Dyspnea of such causation is not infrequent in Hodgkin's disease. A recent article[15] describes such a patient in whom dyspnea and severe asthmatic symptoms supervened when he lay on his left side. Relief was gained when he turned to the right side. A mass in the mediastinum was present.

French[16] lists the causes of the postmediastinal syndrome as follows: aneurysm, a large heart, new growth, hydrothorax with marked displacement of the heart and enormous distention of the abdomen by ascites, tympanites and large tumors. He states that the chief reason why a very large heart or thoracic aneurysm may produce orthopnea, even when signs of cardiac insufficiency are absent, is that when the patient lies down there is less distance between the sternum and the vertebrae than when he sits up. The cause for the orthopnea is thus mechanical: there is more room to accommodate the abnormal mass when the patient sits up. In some cases he may be able to walk about without distress in the daytime, and yet be unable to lie down at night.

The foregoing reminds one of patients with a large pericardial effusion who automatically assume what is sometimes termed the "position of Allah," more accurately designated by the French as the *signe de la prière mahometane*. It is practically a knee-chest position. There are also those patients with huge hearts who cannot sleep save sitting in a chair, often leaning forward on the back of another chair or on another person in front of them. May not both these conditions be due to the relief obtained by lessening the pressure on the structures back of the heart?



A B

Figure 4. *Radiographs to Show Relation of Heart to Spine.*
A — *at full expiration;* B — *at full inspiration. The subject was a normal boy, aged sixteen years.*

There are still other patients who experience severe dyspnea when they assume some particular position in bed. The condition has been long known and has been recently restudied.[17] It is sometimes know as trepopnea. The particular position which causes severe dyspnea varies with different patients. If trepopnea be due to pressure of an enlarged heart on its bed, or on the structures behind the heart, it is reasonable to assume that the differences in the degree and type of cardiac enlargement might account for the variations in the posture that induces the dyspnea. Thus, the left auricle is enlarged in mitral lesions and the left ventricle in chronic arterial hypertension and lesions of the aortic orifice; sometimes enlargement involves the heart as a whole.

Patients with congestive failure often show engorgement of the pulmonary vessels when exam-

ined radiographically. Following rest and digitalis the lung fields may clear and the patient improve. It is well known that a failing heart is prone to be dilated and that digitalis lessens the dilatation. In such a case the compression of the pulmonary vessels would be relieved, and with removal of the obstruction the pulmonary veins would be expected to cease being engorged.

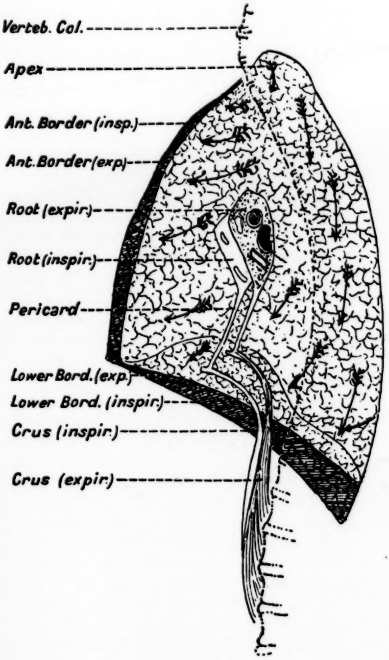


FIGURE 5. *Mediastinal Aspect of the Right Lung to Show the Respiratory Movement of the Hilus.*

The crus of the diaphragm is also indicated, and its attachment to the hilus of the lung through the pericardium. The arrows indicate the direction of the inspiratory movement of the various parts of the lung. (Reproduced from "The Mechanism of Respiration in Man" [Arthur Keith] in Further Advances in Physiology. *[New York: Longmans, Green and Co., 1909.] By courtesy of the publishers.)*

POSTURAL DYSPNEA IN A SERIES OF PATIENTS

A series of cardiac patients have been closely questioned regarding the occurrence of dyspnea while they were in bed; their answers correlated with the fluoroscopic evidence of the backward extension of their hearts. As controls there were selected other patients with proved disease of the heart and usually with some degree of cardiac insufficiency, but whose dyspnea was unrelated to posture.

There were 53 patients in all — 17 with postural dyspnea and 36 without it. The former were all found to have marked extension of the heart backward into the retrocardiac space, whereas but 1 of the control cases had marked encroachment on the retrocardiac space. Some further details will be given, designating the two divisions as Groups 1 and 2.

Group 1. Fourteen patients had the predominant enlargement in the left ventricle; in 2 others it was mainly in the auricles, and in the remaining 1 the enlargement involved both cardiac chambers. Five patients reported that a particular position caused dyspnea more readily — 4 the left lateral and 1 the right lateral position. Two of the 4 left lateral patients found the position impossible to maintain; they were definite examples of the trepopnea described above. Five other patients and 1 of those experiencing trepopnea were subject to paroxysmal attacks of dyspnea at night.

Group 2. All 36 controls had cardiac enlargement. In 17 cases the retrocardiac space was clear; in the remaining 19 the space was encroached on from slight to half its normal area, with 2 exceptions. In one of these the encroachment exceeded an estimated half of the retrocardiac space; in the other it was marked (this patient had a capacious and deep chest). As in Group 1, the encroachment was usually ventricular, the figures being: left ventricle, 16 cases; left auricle, 2 cases; and pericardial effusion, 1 case. One of the group had had postural dyspnea three weeks previously, but it had disappeared at the time of the fluoroscopic examination. Two patients reported that if they lay on the left side they experienced symptoms other than dyspnea; in the first case it was anginal pain; in the second it was described as "grinding and palpitation." Still another patient found a particular position disturbing: that is, anginal pain was prone to appear if he lay too flat.

Subject to the proviso that the number of patients examined for the purpose of correlating postural dyspnea with fluoroscopic evidence of backward extension of the heart was small, certain facts stand out. All patients with postural dyspnea were found to have marked encroachment on the retrocardiac space, usually by the left ventricle, whereas but 1 patient with a similar degree of encroachment failed to experience postural dyspnea. Encroachment on the retrocardiac space to a les-

ser degree — up to half its estimated* area — was common in cardiac patients not subject to postural dyspnea. The detection of 2 cases of definite trepopnea in so small a group was a surprise. Some evidence was also obtained regarding the relative frequency with which a particular posture is more disturbing to the patient. Certain data were not essential to the diagnosis of these cardiac patients and so did not appear in their clinical records, but were obtained when suitable questioning was employed.

Dilatation of the Left Auricle

Some further information was also sought as to whether the back pressure was exerted from the cavity of the left auricle directly into the lumen of the pulmonary veins or by compression of these veins. When the heart is removed from the body at autopsy there are usually one or more sizable holes in the posterior wall of the left auricle. These are clearly an artefact. However, if the inside of this auricle be examined in situ it is found that the pulmonary veins are so arranged as to give some support for the conception that when the auricle is dilated a fold of its wall may close the orifice of the pulmonary veins. A situation may occur that is analogous to that of the urinary bladder, in which distention is said to cause a fold of mucous membrane to seal the urethral outlet and lead to further distention.

It is known that the left auricle is prone to distend, and it is difficult to conceive how this takes place unless the cardiac chamber be subjected to an internal distending pressure. It is likewise difficult to inflate a paper bag if there are holes in it. The capacity of the pulmonary venous system is so great that it is improbable that both the left auricle and the pulmonary veins are distended by blood driven backward, as when the mitral valve is incompetent.

Comment

It is appreciated that the back-pressure theory, based on left ventricular failure, has served to explain much that concerns the heart. The theory offered in this article differs less than one might think at first. The main point is that there is a hindrance to onflow by a mechanical compression of the pulmonary veins by cardiac enlargement. This conception appears more plausible in relation to postural dyspnea. When the patient lies down the structures that in the upright position are posterior to the heart become the bed for it; in other words, the enlarged heart lies on the pulmonary veins and their radicles.

Dyspnea in cardiac patients is prone to be exertional or postural. Figures have been cited in order to emphasize the greatly increased volume of blood flow that takes place in response to exertion, and it is understandable that some obstruction to onflow, such as is present in mitral stenosis, for example, might then prove effective in damming back the flow through the pulmonary veins. An unfavorable posture, such as lying down, might likewise be effective in damming the flow, but might show more variance and develop more slowly depending on the type of cardiac enlargement, and where and to what degree it exerted pressure.

It was believed at first that encroachment on the retrocardiac space might give all the essential information regarding backward extension of the heart. However, it soon became apparent that examination of this space was merely a guide. There are probably other factors, such as posterior extension of the heart elsewhere than directly toward the spine, the position of the diaphragm (Fig. 6), the amount of mobility in the thoracic wall and the size of the thorax as a whole. The reader is cautioned not to interpret the conception of pressure as being exerted solely on the pulmonary veins just before they reach the auricle, — they are stated to measure but 1.5 cm. in length, — but to understand it as including their radicles, as they bring blood to the hili of the lungs.

Backward enlargement of the heart, particularly the left auricle, is credited with exerting pressure that causes such effects as aphonia, dysphagia and hydrothorax. Why may it not also so press on the pulmonary venous system as to cause engorgement of these veins? That the left auricle may compress even the superior vena cava appears from observations by Cossio and Berconsky[18] made during the catheterization of the right auricle in man. Parenthetically, it may be pointed out that the distention of the cervical veins in their patients was not evidence of failure of the right ventricle and back pressure to the systemic veins; heart failure was absent in the cases studied.

Consideration of the findings in the series of patients studied for the possible correlation of encroachment of cardiac enlargement with the presence of postural dyspnea makes it evident that there is a considerable margin of safety; for more than half of the group not subject to postural dyspnea were found to have encroachment up to half the estimated area of the retrocardiac space, and in 2 cases the degree of encroachment was even greater.

There are undoubtedly factors inducing dyspnea other than engorgement of the pulmonary venous system; I refer to various reflexes of nervous origin

*I am responsible for these estimates. I acknowledge the assistance of the roentgenologists of the hospital.

and chemical changes operating on the respiratory center. These are adequately discussed in the textbooks of physiology. The purpose of the present article is to consider the significance of backward encroachment of an enlarged heart. En-

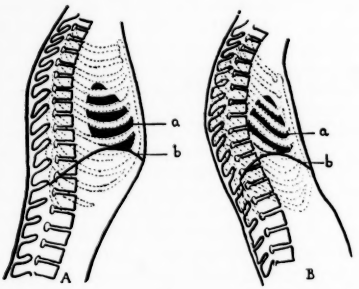


FIGURE 6. *Diagrams Depicting the Effect of the Position of the Diaphragm and the Shape of the Thorax on the Relation of the Heart to the Vertebral Column. (Reproduced from* Body Mechanics in the Study and Treatment of Disease. *J. E. Goldthwait; L. T. Brown; L. T. Swaim, and J. G. Kuhns. [Philadelphia: J. B. Lippincott Co., 1934.] By courtesy of the publisher.)*

gorgement of the pulmonary veins, it is true, has been shown[19] to induce dyspnea through a nerve reflex.

It is not asserted that the contents of this article have proved that engorgement of the pulmonary veins is caused by mechanical pressure exerted on their walls by backward extension of an enlarged heart; this is very difficult to prove. However, it seems reasonable to suggest that physicians should not limit their thoughts to the heart in cardiac patients affected by dyspnea.

What can be done to improve the heart-lung relation? For in a sense are we not all "heart-lung preparations"? Some therapeutic possibilities come to mind. For example, should we not pay more attention to the posture of the patient? The use of a bed rest is a common procedure, but in patients who spontaneously assume the leaning forward posture should we not assist them, and make the position more satisfactory by the use of a pillow laid on a suitably placed table or chair? There are occasional patients in whom elevation on a bed rest gives but partial relief to dyspnea, but who will obtain greater ease if a leaning forward posture is prescribed. I have recently adjusted a hospital bed table in front of such patients and found that it lessened their dyspnea.

Crowding of the available space in the thorax by accumulations of fluid is an evident reason for increased dyspnea. Removal of such effusions would seem to be indicated. An increased application of and a readiness to repeat thoracentesis have appeared to increase the comfort of our patients.

It seems reasonable to suggest the performance of the Brauer operation — the removal of the ribs over the precordia — in patients with marked enlargement of the heart and postural dyspnea, if the latter is unrelieved by medical therapy. The operation is believed[20] to be practically without danger, and can usually be performed under local anesthesia. It has been followed by great relief in 3 cases — 1 reported by Morison[21] in 1909 and 2 by Graham[22] in 1929. Perhaps the significance of these operations has been lost sight of, since the Brauer operation was originally proposed for the relief of pericardial adhesions, although in these 3 cases it was performed for the purpose of decompressing an enlarged heart. I believe this operation to deserve further trial for the relief of the heart-lung field.

SUMMARY

The conception is advanced that backward enlargement of the heart may cause mechanical compression of the pulmonary venous field and lead to engorgement of these vessels.

There are similarities to what is known as the posterior mediastinal syndrome.

Correlation of the fluoroscopic findings with the presence or absence of postural dyspnea resulted in considerable support for the above conception.

The production of engorgement of the pulmonary venous field by the compression caused by backward extension of an enlarged heart appears to explain postural dyspnea better than does the rather generally accepted theory of left ventricular failure.

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REPORT ON MEDICAL PROGRESS

GASTROENTEROLOGY

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THE literature for 1939 concerning the gastrointestinal tract is so voluminous that it is obvious that only a portion of it should be included in this review. For that reason, the present article will concern itself solely with reports of various diseases of the small intestine and of the colon. In certain instances comments and discussions of various articles involve a consideration of surgical procedures. Gastrointestinal disease, however, can rarely be classified as purely medical or surgical, and it is the intention of the reviewer to emphasize the diagnostic and broader therapeutic aspects of the subject matter to be surveyed, without any particular consideration of detailed technical procedures.

SMALL INTESTINE

Although no single pathologic condition involving the small bowel, with the exception of duodenal ulcer, is of great numerical importance, the aggregate of all conditions involving the duodenum, jejunum and ileum is of real clinical moment. With the advent of more precise methods of studying small-bowel physiology and improved x-ray technic, it is quite justifiable to comment on the various conditions involving this portion of the digestive tract. It is important to recognize that diagnosis of small-bowel disease primarily depends on the ability of the clinician to suspect its existence, inasmuch as routine roentgenography rarely demonstrates isolated or even at times fairly widely distributed lesions of the jejunum or ileum. It should be stressed, therefore, that any history of abdominal distress or discomfort that tends to localize itself in the region of the umbilicus should raise the suspicion of disease of the small intestine, and should afford the basis for systematic x-ray studies of this region by repeated fluoroscopic and film examinations, made at hourly intervals.

Megaduodenum is a condition that should be diagnosed only after careful study and only with an exact knowledge of the typical x-ray findings. Sturtevant[1] describes in detail the x-ray criteria for such a diagnosis. Duodenal antiperistalsis, duodenal stasis, violent duodenal peristalsis and the "writhing duodenum" are the x-ray findings to be encountered in this condition, which, as a rule, is of little clinical significance. A discussion of the symptoms that sometimes occur can be found in an article by Weiss,[2] who considers the etiology of megaduodenum and reports 6 cases occurring in members of the same family during three generations. He correctly comments on the fact that the condition may be asymptomatic and quite rightly stresses the importance of very conservative therapeutic measures.

Duodenal diverticula are not uncommon and are almost always not the cause of symptoms. That they may occasionally cause symptoms suggestive of ulcer is indicated in the report by Boland,[3] who describes a death due to an acute perforation of a duodenal diverticulum which occurred at the usual site, namely the second portion of the duodenum. Another report of somewhat similar nature is that by Schunk,[4] who describes the traumatic rupture of a jejunal diverticulum. It should be stressed, however, that as a rule such diverticula cause no symptoms and do not warrant surgical treatment.

An unusual accident is that mentioned by Roach,[5] who describes a fatal hematemesis due to a rupture of an abdominal aortic aneurysm into the third part of the duodenum. The history in this case was suggestive of an old duodenal ulcer, with symptoms extending over a period of ten years and even including one attack

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of bloody vomiting. The patient finally entered the hospital, and as on previous occasions, x-ray films demonstrated a deformity in the region of the duodenal cap, apparently due to external pressure. A sudden fatal hemorrhage occurred, and autopsy revealed no duodenal ulcer but an erosion through the duodenum from a contiguous dissecting aneurysm of the abdominal aorta.

Disturbances of the jejunum and ileum are caused by a variety of diseases. A satisfactory and somewhat routine summary of these causes can be found in an article by Kiefer.[6] Inasmuch as most of the symptoms of small-bowel disease are associated with the existence of partial obstruction, it is of importance to refer to articles dealing with various possible causes. One interesting cause is that reported by Havens,[7] who discusses a case of intestinal obstruction caused by the use of colloidal aluminum hydroxide. This patient died of intestinal obstruction following a seven-day treatment of constant drip of this preparation. Such a serious outcome is rarely to be anticipated, but the frequent use of aluminum hydroxide gel as a treatment of ulcer warrants the suggestion that the preparation should be used with a good deal of care because of its tendency to provide the source for a nonabsorbable solid mass. Adamson and Hild[8] also report a case of small-bowel obstruction due to a mass, in this case occurring in an infant thirty-six hours after birth, the ileum being found to be packed with inspissated waxy meconium. Although the condition is rare, 22 cases have already been reported.

Intestinal obstruction due to intraluminal foreign bodies is really not uncommon, and the article by Storck, Rothschild and Ochsner[9] is of value inasmuch as it reviews a total of 875 cases, exclusive of hernia, neoplasm and peritonitis. In this group, 51 cases were due to foreign bodies, of which the commonest were fecaliths and the next commonest, masses of intestinal parasites. The incidence of obstruction due to intestinal parasites naturally varies in different sections of the country, but the fact that they may cause intussusception, intestinal spasm and perforation of the bowel is worthy of emphasis. The article is complete and well worth careful reading.

Gallstones have long been known to be the source of intestinal obstruction, and the report of Wakefield, Vickers and Walters,[10] although presenting no new material, is of interest in this connection. The authors point out that in every case the gallstones appear to have reached the intestinal tract via a cholecystoenteric fistula. The fistula may not remain patent after the passage of the gallstones. It is significant that some of the patients described had recurring attacks of intestinal obstruction prior to the onset of complete ileus. McQueeney[11] records 2 rather unusual cases in which the patients survived gallstone obstruction of the bowel and lived with persistent intestinal biliary fistulas for fifteen and nineteen years respectively.

A rare benign cause of intestinal obstruction is that reported by Pachman,[12] who adds 3 cases to 33 others already reported in the literature as due to enterogenous intramural cysts. In his cases, the intestinal cysts were the cause of obstructive symptoms and occurred in children under one year of age. Shaw[13] reports 5 cases of polyposis of the small intestine, a relatively rare clinical entity but important because of the tendency to malignant degeneration.

Lieber, Stewart and Lund[14] reviewed the literature on carcinoma of the prepapillary portion of the duodenum in a study of 222 cases, including 17 of their own. This very dangerous condition is uncommon, but it is necessary to point out that in most of the cases reported inadequate x-ray studies had been performed, with the result that only 17 per cent were correctly diagnosed prior to operation. The operative mortality in this condition is extremely high, with jaundice occurring in practically all cases, pain in about two thirds and fever in about one third. An excellent summary of the subject of tumors of the small intestine is that by Cohn, Landy and Richter.[15] It contains an excellent bibliography, with a good review of the literature. The frequency of small-bowel malignancy varies in different statistics, and the subject is also well covered by Medinger,[16] who reviews the cases from the New England Deaconess and the Palmer Memorial hospitals. According to Medinger, malignant tumors of the duodenum and ileum appear to be slightly more frequent than those of the jejunum. Carcinoma occurs most frequently in the duodenum and jejunum, and sarcoma in the ileum.

Leiomyosarcoma is extremely rare, only 6 cases being mentioned in the literature, 1 a recent case reported by Foshee and McBride.[17] Another form of tumor that is uncommon but worthy of comment is the argentaffine tumor, which for the most part is found in the region of the appendix. Several individual cases are reported, but the series of 72 cases mentioned by Porter and Whelan[18] is of particular interest. The incidence of the tumor can be readily seen in that only 72 cases were found in a total of 26,000 surgical specimens examined. In addition to these tumors in the appendix, 2 were found in the stomach, 1 in the gall bladder, 1 in the duodenum and 8 in the

small bowel. None of the appendiceal tumors were malignant, but of the 8 argentaffine tumors of the small intestine, 3 were malignant.

The importance of Meckel's diverticulum as a cause of intestinal obstruction is well known, as is the fact that not infrequently the diverticulum contains gastric mucous membrane. The case report by Martin[19] describing an intussusception from such a diverticulum contains nothing new, but the accompanying résumé of the literature is presented in a well-organized fashion and is an unusually complete description of this form of small-bowel disease. The discussion includes a consideration of the condition, the acute and inflammatory process associated with Meckel's diverticulum, ulceration, perforation, hemorrhage and obstruction.

Hyperplastic tuberculosis of the small bowel is a recognized but rare cause of obstruction. Because of its infrequency, it is not generally recognized as a possible explanation for symptoms of intestinal obstruction, and for this reason the report by Caplan and Roantree[20] is worthy of consideration. Mesenteric adenopathy is well known to be a cause of symptoms simulating appendicitis or partial obstruction of the ileum, and may be a manifestation of tuberculous nodes or of some nonspecific inflammatory involvement. A complete description of the abdominal syndromes from adenopathies of the mesentery is presented by Parini,[21] who discusses differential diagnosis and very properly concludes that surgical interference is usually indicated only in acute forms in the presence of intestinal obstruction due to acute suppuration of the nodes. Too frequently, the demonstration of calcified lymph nodes throughout the mesentery is taken as an adequate reason for surgical intervention, although it not infrequently happens that symptoms are due to other causes or are unrelieved by surgical procedures.

An unusual form of intestinal obstruction is that reported by Orr,[22] who records 2 cases associated with mechanical injury to the spine. The difficulties of diagnosis are obviously increased with a cast covering the entire abdomen, the pain being attributed in one of the cases to a broken back, and in the other to a paralytic ileus and pressure of the cast. In neither case was the diagnosis of intestinal obstruction adequately considered.

Intestinal intussusception in infants as a cause of small-bowel obstruction is well recognized. The report of Garcia[23] on 29 such cases is of interest, largely as a review of the physical findings and the technical surgical procedures necessary to treatment. Intussusception in adults is less common, however, and for this reason the short report of Miller[24] is of interest as indicating that it may occur in the presence of benign tumors of the small bowel or in relation to Meckel's diverticulum. A somewhat similar report by Mendelson and Sherman[25] also emphasizes the various causes that may lead to intussusception of the small bowel or, for that matter, of the large bowel.

In discussing acute obstruction of the small bowel, Holt[26] emphasizes the obvious necessity of early diagnosis and treatment. Because of the appearance of the irreversible circulatory changes that take place, he makes out a good case for the therapeutic value of intestinal decompression and subcutaneous salt solution prior to actual surgery for the condition. A more valuable discussion is that of Gendel, Fine and Rosenfeld,[27] who record the results of experimental acute intestinal obstruction in dogs on blood plasma volumes. These authors stress the importance of the early progressive loss of blood plasma, which may reach a figure of 36 per cent within four to six hours, with a loss of plasma volume of as high as 55 per cent within twenty-four hours after the production of the obstruction. Such a loss of plasma, which is equivalent to around 3 per cent of the body weight, is more than sufficient in itself to cause death, and occurs particularly in those cases in which strangulation is added to obstruction. Blood-pressure readings apparently are not so accurate a guide to the patient's condition as are the hematocrit readings with their direct evidence of the loss of plasma volume. The administration of adequate quantities of plasma by the intravenous route is recommended by these authors, with sound reasons for advocating such a measure.

Appendicitis

That appendicitis still presents a challenge to the medical profession is obvious from the number of articles that have appeared during the past year on this subject. An increasing mortality from this condition emphasizes the necessity of educating the public against the continuous use of cathartics for abdominal pain and against delay in the diagnosis and treatment of acute abdominal pain, not only in the right side but also in the middle of the abdomen. This fact has been properly stressed by a rather forceful letter[28] in the *Journal of the American Medical Association*. The seriousness of the problem is apparent from the fact that approximately 16,000 deaths are reported in this country every year as due to acute appendiceal peritonitis. In Canada, appendicitis rates tenth as a cause of death.

The danger of delayed diagnosis is emphasized in an article by Kelly and Watkins,[29] who present

an interesting review of the results of 1000 consecutive cases of acute, simple and chronic appendicitis operated on within recent years, and contrast these with the results obtained in 1000 similar cases reported by them in 1931. The results in the latter series show no great variation from those previously reported, except for a sharp increase in the mortality rate noted in those classified as acute suppurative appendicitis. Under this classification there was a rise from a 9 per cent mortality in the first series to 23 per cent mortality in the second group. A similar increase in deaths from acute appendicitis was noted in an entirely analogous report by Quain and Waldschmidt[30, 31] on two 1000-case studies reported in 1928 and 1934 respectively. They reported a mortality of 18.8 per cent during the depression years, as compared with a mortality of 10.0 per cent prior to that time. Kelly and Watkins believe that this rather alarming increase in mortality is due to a delay in operating because of economic reasons during the depression years and a tendency to use home remedies, including purgatives, before calling a physician. Meyer and his collaborators[32] show about the same mortality rate at the Cook County Hospital in the years 1937 and 1938 as compared with the period from 1928 to 1932.

Numerous reports, such as those of Munroe,[33] Horsley,[34] Raffl[35] and Lundgren, Garside and Boice[36] seem to indicate that the mortality from certain groups of uncomplicated cases may be as low as 0.7 per cent. Perusal of these case reports, however, suggests the possibility that these low mortality figures were encountered in the most favorable age groups, exclusive of children and older people, and quite probably in patients of the higher economic levels. Obviously, increased mortality rates are found in cases where delay has occurred and peritonitis is a complicating factor.

The treatment of over 100 consecutive appendectomies complicated by local or generalized peritonitis with non-drainage as recorded by Pickell,[37] with a mortality of only 4 per cent, suggests that unusually great care was observed in the preparation and handling of individual patients. Some of the factors that may help lower the mortality are suggested by Wright, Aaron, Regan and Milch,[38] who report two small series of cases treated in 1935 and from 1935 to 1939, respectively. In all the cases there was diffuse peritonitis following perforation of the appendix. In the first group, the mortality was 45 per cent, in the second group, 12 per cent. In the first group the patients were operated on by six different surgeons, with no uniformity of treatment. In the second group three surgeons were responsible for the care of the patients, and in their 60 cases it was concluded that the intelligent use of morphine, fluids, duodenal decompression and transfusions were of great effect in combating the hazard of peritonitis, with subsequent lowering of mortality.

The value of decompression by the Wangensteen technic is stressed by various authors, particularly where perforation has occurred. In children the need of immediate operation once the diagnosis has been established is stressed by numerous writers. In the uncomplicated cases, all the writers agree that the mortality is low. Thompson[39] discusses a group of 110 children with extra-appendiceal extension, and expresses the belief that the chief factor in bringing about recovery is the nearly complete removal of fluid and gas from the distended paralytic or obstructed bowel loops. By careful attention to detailed preoperative measures, mortality was limited to 4.5 per cent in a group of 110 seriously ill children.

The importance of deferring operation in the presence of peritonitis due to ruptured appendix in children is discussed by Elman.[40] Decision to postpone operation, he thinks, depends not so much on the presence or absence or the degree of peritonitis as on the general condition of the patient. The amount of clinical toxicity obviously influences the mortality to a marked extent. In those patients with perforated appendices who were operated on at once, the mortality was extremely high, whereas in those operated on after careful treatment and a delay of from nine to twenty-four hours, the mortality was less than half as great. On the other hand, in the nontoxic group the condition was reversed, delay in operating apparently causing 15.0 per cent mortality as opposed to a mortality of only 3.5 per cent in those children operated on immediately. From these articles and from numerous others, it is obvious that most of the factors determining mortality are well known. Even in the most serious cases, however, sufficient delay with adequate preoperative treatment, including decompression of the distended small bowel and the proper use of fluids, tends to reduce deaths to a fairly reasonable figure. In passing, it is of some interest to mention the case reported by Redon,[41] if only to illustrate that at times appendicitis or acute intussusception may be completely reproduced by an allergic disturbance (Quincke's edema), with edema of the bowel. As a rule, the diagnosis of appendicitis is simple, but such a diagnosis is not always warranted unless a fairly complete history has been obtained, which should include a few questions as to allergic disturbances.

The seriousness of "laxative induced peritonitis"

is brought out in an article by Bower, Burns and Mengle.[42] In this report the authors discuss the use of a concentrated *Clostridium welchii* antitoxin preoperatively or postoperatively as a means of localizing peritonitis. Unfortunately no control figures are given, but the report is of theoretical and possibly of practical interest. In this connection the paper by Bain and Feagles[43] is also of some interest. These authors treated 100 cases of peritonitis from ruptured appendices and 15 other cases due to perforated viscera with intra-abdominal irrigations of peptone broth. Apparently very favorable results were obtained, with a drop in temperature and pulse and a reduction in pus drainage. The exact mechanism of such irrigations is discussed in terms of stimulation of the body-defense mechanism by foreign protein or stimulation of the formation of bacteriophage. Such a maneuver is obviously in an experimental stage, but is of interest in relation to the work of Steinberg and others.

Of further interest is the report by Carry, Brewer and Nicol,[44] who record the postoperative treatment of appendiceal peritonitis with sulfanilamide. Twenty-six patients with general peritonitis and 15 cases of appendiceal abscess were treated postoperatively with sulfanilamide, with 3 deaths. Of the patients who died, only 1 at necropsy still had general peritonitis of the lower abdomen. Although the number of cases reported is too small to be statistically important, it will not be surprising if sulfanilamide or one of its derivatives proves of real worth in the management of these badly infected cases.

An unusual complication of appendicitis is that reported by Parker.[45] The patient was operated on for chronic appendicitis, and recovery was uneventful. Three weeks after leaving the hospital the patient returned with a complaint of a rectal hemorrhage following a bowel movement. Sigmoidoscopy was negative. Two subsequent hemorrhages occurred, the last one of serious proportions. Exploration revealed active inflammation of the cecum in the region of the stump of the appendix and a mass within the cecal wall, which on exploration was found to be an abscess cavity that had eroded into a large artery.

Ulcerative Colitis

Chronic idiopathic ulcerative colitis still remains an unsolved problem of great importance, and although studies as to its etiology are numerous, they are still unconvincing. Two articles by Reed[46] present in summarized form the various views that have attracted attention as to the causation of this very grave condition. Reed records the observations of Hurst, Thorlahson, Felson and others on the relation between this condition and bacillary dysentery, and also considers the well-known studies of Paulson, Bargen and Dack. His own bacteriological studies are in accord with those of Paulson, who has concluded that as yet the disease has not been shown to be due to any demonstrable single infectious agent. He stresses the need of considering bacillary dysentery, amebic dysentery and also other factors that are incident to the disease, such as psychogenic disturbances, deficiency states due to malabsorption and so forth. He also points out the difficulty of accurate timing of surgical intervention, a fact that is well worth emphasizing. The articles contain nothing new but are of some interest because the author reiterates most of the considerations that are generally accepted in relation to ulcerative colitis.

One of the most significant contributions to an understanding of the subject is found in the brilliant observations of Lium.[47] In experiments on dogs, by means of colonic implants he was able to demonstrate that mechanical stimulation, parasympathetico-comimetic drugs or dysentery toxin causes spasm of the colonic musculature, with resulting damage to the underlying epithelial structure, hemorrhage and ulceration. The mode of action of dysentery toxin in producing ulcerations of the mucosa is thus apparently due to the injurious effects of smooth-muscle spasm. Hypersecretion of mucus was associated with increased muscular contractions at first, but later a thin watery secretion, inadequate for protective purposes, was observed, with resulting inadequate protection of the mucous membranes from trauma. Lium discusses the possibility that ulcerative colitis may be conceived as a specific reaction to a number of influences that can initiate spasm of the colonic musculature. These include possible hyperactivity of the parasympathetic nervous system, infections, such as bacillary dysentery, and vitamin deficiency. Once the colon becomes spastic, it is potentially an organ that can produce damage to its own surface structures. In analyzing the distribution of lesions observed in 6 fatal cases, Lium and Porter[48] conclude: "It seems highly probable that ulcerative colitis is primarily a disease caused by intense muscular spasm and hypermotility and that, therefore, the distribution of the lesions follows a muscular pattern. Whatever infectious element is present may well be due to secondary involvement of damaged areas caused by muscular overactivity." It is not difficult to draw an inference from a comparison between these striking observations of Lium and Porter and many of those cases of ulcerative colitis that appear to have their fundamental origin in an unstable and over-

stimulated autonomic nervous system. The changes visualized by Lium following the action of parasympathetic drugs are not dissimilar to those reported by White and Jones[49] and bear a direct relation to the clinical observations of Sullivan,[50] Murray[51] and Alexander[52] on the importance of emotional factors in initiating this disease.

Clinical observations by Lium[53] on 8 normal individuals and 1 patient with ulcerative colitis are of interest in this connection. Measurements were made of contractility under measurable stimuli of the rectum, and the resulting rectometrograms indicated that the rectal contractions were much more powerful and prolonged in the presence of ulcerative colitis and were less amenable to relaxation by spinal anesthesia than in normal people. The importance of these continued contractions in the chronicity of ulcerative colitis is discussed. In this regard the studies of Bargen and Jackman[54] are of interest therapeutically. They studied the influence of papaverine on the muscular tone of the intestinal tract. In their hands, papaverine, alone or combined with pantopon, has seemed to be of value in relieving abdominal cramps and frequent purulent bloody discharge in cases of colitis. The evidence obtained experimentally substantiates the antispasmodic effect of papaverine, and it appears to have certain advantages over pantopon in cases where it is desired to immobilize the intestine or put it to rest.

Specific measures for the medical treatment of this disease continue to accumulate. For the most part they have to do with local treatment of the bowel, treatment of various deficiency conditions resulting from the disease and chemotherapeutic measures directed toward more or less control of the factor of bacterial infestation. Gainsborough[55] treated a group of patients with cod-liver oil retention enemas, and while he frankly states that the method was certainly not a curative one, he believes that it was very efficacious in shortening the illness and reducing pain. He points out that these patients were more tolerant to cod-liver oil enemas than to other local applications, a possible point of value in what is obviously merely palliative treatment.

Lerner and Rapaport[56] assert that vitamin A deficiency is one of the complications of ulcerative colitis, a statement which is not surprising in view of the probable interference of the disease with all fat-soluble vitamins. Such a finding indicates, however, that deficiencies secondary to ulcerative colitis are numerous, and illustrates the complexity of the body changes incident to the disease. The suggestion of Cheney[57] that intensive liver therapy may modify the course of the disease is of some interest, but the evidence that he presents is unconvincing. There is little doubt that satisfactory remissions accompanied the administration of liver extract, but one must remember that similar remissions occur spontaneously and frequently in this disease. It may well be that liver therapy does improve the patient's general condition to a high degree, but it is doubtful that anything further than this is accomplished.

Although no single chemotherapeutic measure has as yet been evolved that controls the relapses in this condition, sulfanilamide, Neoprontosil and other drugs have been mentioned by various writers. One of the most recent reports is that of Hebb, Sullivan and Felton,[58] who discuss the results obtained in the treatment of 14 cases with rectal lesions of lymphopathia venereum and 4 cases of chronic ulcerative colitis with sodium sulfanilyl sulfanilate or with sodium sulfanilate. That the series is altogether too small to warrant definite conclusions is obvious and is admitted by the authors. The improvement observed in both groups, however, is of interest, and in considering the patients with ulcerative colitis, one cannot help but hope that some such measure may control, at least to some degree, what obviously is frequently a bacterial infection of a badly damaged bowel. Any such help would be a welcome addition to our therapeutic measures in controlling the violent and dangerous exacerbations of the disease.

The surgery of the condition has been reviewed by various authors, and it is generally accepted that with the proper indications, an ileostomy, with or without a colectomy, is the best available method at present. The report by Cave,[59] while not presenting any new material, is of interest in this respect.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 26151

PRESENTATION OF CASE

A thirty-eight-year-old housewife was admitted to the hospital complaining of excruciating abdominal pain, with vomiting of five hours' duration.

On retiring at midnight after a somewhat lively family birthday celebration the patient was aware of epigastric discomfort. Considering the party, this was interpreted as the result of natural causes, and she went to sleep. She awoke, however, about 3:30 a.m. with an intense upper abdominal pain, and began to vomit repeatedly. The pain was constant and unrelenting and felt like a "hot lead pipe the length of the abdomen." It radiated to the back and gradually extended downward. There were no cramps or periumbilical pains. The patient writhed and tossed in the bed and refused to call a physician; at 5:30 a.m. the husband finally called one. The doctor examined her thirty minutes later and found a "silent" abdomen which was somewhat rigid, especially in its upper portions; he also observed flecks of bright and changed blood in the vomitus. She was immediately referred to the hospital.

The patient had been married eighteen months, but had not been pregnant. Six months before admission she was told that she had a "fibroid." Several years before entry she had had an appendectomy in an outside hospital.

Physical examination at 8:00 a.m., or about five hours after the onset of the acute pain, revealed a well-developed and well-nourished woman who did not appear very sick. Examination of the heart and lungs was negative. There was no audible peristalsis, and the abdomen was neither distended nor showed areas of definite tenderness; both rectus muscles were held tight while the sides of the abdomen were soft. Pelvic examination revealed the presence of a tense, tender, cystic mass occupying and filling the posterior cul de sac; the patient identified this mass as the source of her pain. The remainder of the examination was negative.

The temperature was 98°F., the pulse 85, and the respirations 20.

Examination of the blood showed a white-cell count of 10,800, and that of the urine was negative.

Plain roentgen films of the abdomen taken in the supine, upright and laterosupine positions showed no definitely abnormal loops of small bowel. In the upright position there was a fluid level to the right of the spine in the region of the gall bladder. Since the patient had had an enema before the film was taken, it was thought that this fluid level could have been in the colon instead of the small bowel. There was a small quantity of gas in a nondilated loop of small bowel at the level of the fourth lumbar vertebra. The soft tissues in the pelvis were dense, and there appeared to be a rounded smooth mass in the region of the uterus. There was no air under the diaphragm.

After catheterization the pelvic findings were unchanged. She was taken to the operating room, and an exploratory laparotomy was immediately performed.

DIFFERENTIAL DIAGNOSIS

DR. ARTHUR W. ALLEN: May we see the x-ray films?

DR. FELIX FLEISCHNER: The diaphragms are somewhat high, but there is no free gas below either leaf. I can see the fluid level mentioned. The amount of gas in the bowel appears to be about normal. However there is an area in the lower abdomen and pelvis which fails to show any gas-filled intestinal loop. This coincides with the soft-tissue mass which was described. We can rule out perforation of a viscus.

DR. ALLEN: Would you say that the elevation of the diaphragm would give any information about the intra-abdominal pressure?

DR. FLEISCHNER: No; I cannot even state that the position of the diaphragm is unusually high.

DR. ALLEN: We have a young woman with a previously diagnosed pelvic mass, thought to be a fibroid, with a sudden attack of abdominal pain. The pain was very excruciating, coming on suddenly and radiating to the back without any cramps and with a normal temperature and pulse and a slightly elevated white count five hours after onset. The x-ray films showed a soft-tissue mass in the region of the pelvis. The pelvic examination before operation described a tender cystic mass in the pelvis. All these findings lead up to a perfectly classical textbook story of ovarian cyst with a twisted pedicle. Twists of the ovary and fallopian tube, of pedunculated fibroids and so forth are apt to be sudden in onset and to come on while the patient is straining at stool. Such patients do not have any rise in temperature until gangrene develops in the obstructed organ, and an elevated white-cell count is the first laboratory evidence that there is something serious the matter. It is usually around 11,000 at the end of five hours, and it gradually rises.

There are certain factors about this case that

make me slightly suspicious that this obvious diagnosis is not correct. In the first place we are not sure what she means by "pain radiating to the back." If the patient had a penetrating ulcer of the posterior wall of the stomach or duodenum into the pancreas, the pain should radiate directly through the back at that level. If the diseased organ is the gall bladder and the pain radiates to the back the pain may be immediately behind the gall bladder but is more apt to be in the subscapular region. In a patient with small-bowel obstruction one of the chief complaints is backache that is usually in the lumbar region. If the patient has a pelvic disorder with backache, the pain is still lower,—down over the sacrum,—and that holds also for low large-bowel tumors, such as tumors of the sigmoid and rectum. We are only told that this pain radiated to the back.

One of the important observations is the fact that the vomitus was flecked with blood, but I think it probably has no significance because this patient obviously vomited very hard and the flecks of blood probably came from the engorged mucosa, either of the pharynx or esophagus. I believe that the physician who saw her first might have thought that the "silent," tense abdomen, the sudden onset of pain and the liquid indiscretions of the preceding evening might all have contributed to a perforated peptic ulcer. I think it is quite likely that was the provisional diagnosis when she came in here. The physical signs on admission seem to have ruled that out, and I believe the x-ray department believes that air will be found under the diaphragm in 80 to 90 per cent of cases with perforation of a hollow viscus; none was found here. The previous history of indigestion does not mean anything because a silent ulcer may perforate and bleed. I think we can rule out this possibility and that of any disease in the upper abdomen reasonably well.

There is one important part of the history that we must pay very strict attention to and not forget for a moment; that is, the fact that she had had an appendectomy a few years before. One of the common causes of subacute abdominal pain is intestinal obstruction on the basis of adhesions; it may or may not be preceded by a previous operation, but is more apt to come about if an operation has been performed.

Could this be a case of small-bowel obstruction? The onset of the pain was consistent with such a diagnosis, but I should expect the pain to have been described as crampy and not constant, and it states very definitely that there were no cramps or periumbilical pain. The fact that there was a cystic mass in the pelvis does not rule out intestinal obstruction, because we have frequently found during phases of ileus or obstruction a dilated loop of small intestine actually filling the pelvis and feeling like a cyst on pelvic examination. Frequently such obstructed loops of bowel have been mistaken for abscesses in the pelvis. The outs about trying to tie this up with small-bowel obstruction are quite numerous. The character of the pain was wrong. We note the radiation of pain, but not to the right portion of the back. The fact that her abdomen was "silent" at the time the physician first listened to it before she had medication and again when she came in here is also against it. Also, I think it is unusual in intestinal obstruction not to have more evidence of dilated loops by x-ray even at the end of five hours, and I should not expect such a clear-cut round shadow as we have in this case.

I think one has to come down to an acute abdominal condition on the basis of a pelvic mass that has not gone far enough in its process to produce fever or much change in pulse or respiration, and only a slight elevation in white-cell count. I have to come back to my first impression, namely an ovarian cyst with a twisted pedicle.

DR. WALTER BAUER: Might it not have been an acute gastritis and an ovarian cyst which was not twisted?

DR. ALLEN: I think that is quite possible. I should hardly expect the pain to persist after she stopped vomiting and to be associated with this other mass which was felt by pelvic examination.

DR. HORATIO ROGERS: Dr. Richardson saw this patient first, and I saw her when she came into the hospital. At first, as Dr. Allen discussed it, I thought he must be familiar with the case because he followed exactly our line of reasoning. Dr. Richardson at first thought it was probably an ulcer which had perforated into the lesser peritoneal cavity, and after we had the patient in the hospital and had more data my diagnosis was twisted ovarian cyst. It was on that diagnosis that I operated and found 50 cm. of gangrenous small intestine in the pelvis, free bloody fluid and a tight band of omentum adherent to the site of the appendectomy under which the intestine had gone. It was quite easy to put my finger under this band, lift it up and cut it, whereupon the gangrenous intestine came up into the wound. We could then see the constrictions where the bowel and the mesentery had been dragged through under the band. We watched the intestine a reasonable length of time for the return of color, but as it stayed black it was resected. The patient made an uneventful recovery and went home on the sixteenth postoperative day.

DR. TRACY B. MALLORY: Have you anything to add, Dr. Richardson?

DR. WYMAN RICHARDSON: My diagnosis was

ruptured gastric ulcer. When I heard about the pelvic examination I said: "I did not do it. Perhaps there was pus down there in the pelvis." The first striking thing about this patient was the subnormal temperature; furthermore, she seemed to be in good condition in spite of the most terrific pain. Another interesting feature was that the pain at six o'clock in the morning was high above the umbilicus in the epigastrium, radiating straight through to the back; it was very severe and non-cramplike. Later it began to radiate down in a vertical line, never radiating horizontally across the back. In retrospect I think probably the last is an important clinical finding to emphasize.

DR. MALLORY: Did you find a fibroid?

DR. ROGERS: Yes; there was an 8-cm. fibroid which had nothing to do with the picture.

DR. BAUER: Do you think that what you felt was the loop of small bowel?

DR. ROGERS: I am certain of it. It was impressive how accurately the patient could tell when I touched it. It is of interest that when I reviewed the patient's history with her a few days later I still could not elicit a story that sounded at all like that of intestinal obstruction.

DR. BAUER: If you were asked to feel a pelvis like this again, would you be able to say: "This is small bowel and not a cyst"?

DR. ROGERS: No; I think I should make the same mistake again.

DR. GEORGE W. HOLMES: How long before the operation was the x-ray taken?

DR. ROGERS: About five and a half hours.

DR. HOLMES: The absence of dilated loops in the bowel is difficult to explain. Have you any explanation?

DR. ROGERS: She apparently did not have time to develop gaseous distention. The loops of bowel in the pelvis were not distended with gas but with fluid.

CLINICAL DIAGNOSIS

Twisted ovarian cyst, with strangulation and gangrene.

DR. ALLEN'S DIAGNOSIS

Twisted ovarian cyst.

ANATOMICAL DIAGNOSIS

Gangrene of small bowel secondary to strangulation by adhesive band.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The specimen we received showed gangrene of the bowel, but we were disturbed by the fact that there was no thrombosis either in the arteries or in the veins. Evidently the strangulation by the adhesive band was so complete that the blood supply was totally cut off.

DR. GRANTLEY W. TAYLOR: Have you any knowledge at what level of small intestine the obstruction occurred?

DR. ROGERS: About 50 cm. above the cecum.

CASE 26152

PRESENTATION OF CASE

An eleven-year-old American schoolboy entered the hospital with the complaint of jaundice of thirteen days' duration.

He had been perfectly well until thirteen days before entry when his schoolmates told him that his eyes were yellow. He had no complaints at that time except slight anorexia, although his urine was dark colored and his stools were somewhat paler than normal. He had no fever, vomiting, diarrhea or definite malaise, but occasionally did feel slightly nauseated. He stayed in bed for three days and at home for a total of a week. He returned to school for three days, after which his vacation began. His condition remained unchanged until two days before entry when it was noticed that his abdomen was enlarging. He began to complain that he had some abdominal pain when he bent over and that he felt somewhat listless. At no time had he had any fever, and the appearance of his jaundice was not preceded by any event which he could remember, such as the eating of a heavy meal. During the evening before entry he had severe pain in his rectum while moving his bowels. He had had no other symptoms.

The past history and family history were essentially negative. He had had measles, whooping cough, mumps and chicken pox. Before entering school his tonsils and adenoids had been removed because of occasional sore throats. He had had two or three colds a year, but never any previous jaundice or prolonged periods of malaise. None of his schoolmates and no other members of his family were ever known to have had jaundice.

Physical examination revealed a well-developed and well-nourished boy in no apparent discomfort. The skin and sclerae were light yellow in color. The heart and lungs were negative. The blood pressure was 120 systolic, 80 diastolic. The abdomen was very protuberant, and the umbilicus bulged. The percussion note was tympanitic in the midline and dull in the flanks. There was shifting dullness, but a definite fluid wave could not be made out. There was questionable tenderness, but no spasm. The liver could be palpated about 3 cm. below the costal margin. Its edge was moderately firm, slightly tender, sharp and definitely

uneven. The left lobe of the liver could not be palpated, but there was a mass in the left upper quadrant extending about 3 cm. below the costal margin, which seemed to move on respiration. The extremities were negative.

The temperature was 99.5°F., the pulse 80, and the respirations 20.

The urine examination was negative. The blood showed a red-cell count of 3,400,000 with 70 per cent hemoglobin, and a white-cell count of 5600 with 62 per cent polymorphonuclears. The stools were brown in color and gave a ++ test for bile. The whole-blood nonprotein nitrogen was 28 mg. per 100 cc., the serum protein 7 gm., with an albumin-globulin ratio of 1.0, the van den Bergh 13.4 mg. biphasic, the phosphorus 2.90 mg., and the total cholesterol 135 mg. The chlorides were equivalent to 114 cc. of N/10 sodium chloride. A sugar-tolerance test showed a fasting level of 54 mg. per 100 cc., 109 mg. at a half hour, 160 mg. at one and a half hours, and 80 mg. at three hours. The blood Hinton test was negative, as was a 1:1000 tuberculin test. An x-ray of the abdomen showed an enlarged liver and spleen. There were no unusual areas of calcification, and the kidneys were normal in size, shape and position. There was no evidence of disease in the bones of the arm or in those around the knee joints, and a skull plate was negative. An x-ray of the esophagus showed it to be slightly dilated. The rugae of the lower half were tortuous, definitely indicating the presence of varices.

For the first month in the hospital the temperature rose daily to 100°F., falling to normal during the night. Several urine examinations were normal, and the stools continued to contain bile. The red-cell count remained at a level of about 3,500,000, but he showed a persistent leukopenia, the white-cell count falling as low as 3250 with 65 per cent polymorphonuclears. During the first ten days the jaundice practically disappeared, but the ascites persisted. His condition otherwise remained unchanged.

An x-ray film of the chest taken at the end of the first month showed excellent excursion of the heart, with no calcification in the pericardium, no widening of the superior mediastinum and no evidence of adhesive pericarditis. On the thirty-seventh day an omentopexy was performed, following which he developed an acute otitis media which drained spontaneously. A culture of the pus yielded hemolytic streptococci. For seven days after the operation he had a septic temperature rising daily to 102°F., and he developed signs of consolidation in the chest. An x-ray film showed hazy dullness in the middle third of the right lung and at the left base. However, the otitis media and apparent pneumonia cleared up completely in a few days.

His course was uneventful for the next three weeks but at that time, nine weeks after entry, he began to have greater elevations of temperature, with a daily rise to 101 or 103°F. By that time the red-cell count had fallen to 2,800,000, with 56 per cent hemoglobin; and the white-cell count was 4600, with 38 per cent polymorphonuclears, 54 per cent lymphocytes and 8 per cent monocytes. A repeat x-ray plate of the esophagus showed varices more clearly visible and wider than at the first examination. A bromsulfalein test of liver function showed 40 per cent retention after five minutes, and 20 per cent after thirty minutes. The serum protein was 6.5 gm. per 100 cc., the van den Bergh 3.41 mg., and the nonprotein nitrogen 23 mg. During the tenth week he again developed jaundice, which disappeared in the next ten days. The ascites increased in amount, and he had some diarrhea.

More careful inquiry into the patient's family history revealed syphilis in the mother and older sister. The mother had had positive serological tests one year before the patient's birth and had had three or four treatments at that time. On the basis of this, antisyphilitic therapy was begun, with potassium iodide and mercury by mouth. A lumbar puncture showed normal findings, with a negative Wassermann test on the spinal fluid. An abdominal paracentesis was done at the end of the twelfth week, and 4000 cc. of clear straw-colored fluid was removed. A Wassermann test on the fluid was moderately positive. In the meantime the temperature fluctuation had become less marked, rising only to 100°F. daily. Two cubic centimeters of Mercupurin intravenously produced 4500 cc. of urine in the following twelve hours; two more abdominal paracenteses were done, yielding a total of 12,300 cc. of straw-colored fluid. In the middle of the fourteenth week the urine for the first time showed a trace of albumin and contained occasional casts. Two subsequent specimens showed similar findings, and one contained 5 to 10 red cells per high-power field.

On the second day of the sixteenth week he suddenly vomited about 500 cc. of coffee-grounds material streaked with red blood. He became very restless and somewhat delirious and cried out as if in pain. There was a definite uremic odor to the breath. The nonprotein nitrogen of the blood serum was 55 mg. per 100 cc., and the serum protein 5.5 gm. He was given ⅛ gr. of morphine subcutaneously for the restlessness, and soon lapsed into coma with Cheyne-Stokes respirations. He remained in coma until his death, thirty-six hours later.

Differential Diagnosis

Dr. John H. Talbott: The first diagnosis which

enters my mind when mention is made of an eleven-year-old jaundiced schoolboy is catarrhal jaundice. As we read through the history, however, it is obvious that the diagnosis is not so easy as this. I should like to stress the point that he felt perfectly well until shortly before admission. The negative family history helps us to exclude certain familial maladies, such as hemolytic jaundice, Gaucher's disease and Cooley's anemia. On physical examination he was well developed and well nourished but had a prominent abdomen and a bulging umbilicus. Dullness in the flanks and the tympanitic percussion note in the midline confirm the impression that there was an increased amount of abdominal fluid. It is possible for a huge spleen, such as may be found in myelogenous leukemia or Gaucher's disease, to simulate ascites, but any such degree of splenomegaly evidently was not present. The only mention that is made of a palpable spleen is the statement that a mass was felt in the left upper quadrant which descended on respiration. Until proved otherwise I shall consider this to be a spleen.

The enlargement of the liver, anemia and leukopenia are the first data which suggest that the malady from which this patient suffered was more chronic than the history admits. The anemia was associated with a color index that was slightly above normal, a fact which indicates that the former was not caused by chronic blood loss. The presence of bile in the urine together with a brown-colored stool is in favor of intrahepatic rather than obstructive jaundice. The concentration of serum protein was normal, namely 7 gm. per 100 cc. However, the albumin-globulin ratio was decreased, undoubtedly due to an increased concentration of serum globulin at the expense of serum albumin. The concentration of cholesterol was 135 mg. per 100 cc. Similar low normal values may be found in patients with an acute hepatitis. The serum chlorides were considerably above normal. I recall one patient suffering from an idiopathic cirrhosis of the liver and bleeding esophageal varices who had a concentration of serum chlorides even greater than this. I do not know the significance of this association. The sugar tolerance curve is consistent with intrahepatic disease. The x-ray studies of the esophagus showed varices in the lower portion. The x-rays of the long bones and skull were taken presumably to help exclude xanthomatosis and Gaucher's disease. During his stay in the hospital further x-rays were taken to detect any calcification of the pericardium. From the data given in this abstract, I doubt very much whether I should have thought seriously of constrictive pericarditis to explain the whole picture.

An omentopexy was performed during the sixth hospital week. I should have been pleased to have a statement from the surgeon concerning the texture of the splenic and portal veins as well as that of the spleen. Following operation he contracted an acute otitis media and, later, patchy consolidation of the lungs, possibly a transient streptococcal pneumonia. Daily elevation of temperature suggests lymphoma or Hodgkin's disease, but there is little else to corroborate this impression. It is true that there was a relative increase in lymphocytes, but I should certainly not make a diagnosis of lymphoblastoma on this one report. The 20 per cent retention of bromsulfalein after thirty minutes is consistent with moderate impairment of liver function.

The family history of syphilis was discovered rather late in the disease to be of much help to the patient or to us in our differential diagnosis. On admission the blood Hinton reaction was negative, and the spinal-fluid Wassermann test subsequently negative. A moderately positive ascitic-fluid Wassermann test only confuses me. If this patient were suffering from congenital syphilis of the liver and spleen, I should expect other stigmas and positive serum reactions.

Albumin and casts were noted in the urine shortly before death. These may be part of what is called the hepatorenal syndrome, about which, I confess, I have only a very hazy mental picture. The exitus was a rather precipitous one. The patient had a massive hemorrhage and vomited old and new blood. The restlessness following this hemorrhage or hemorrhages was treated with morphine. Apparently whoever ordered this drug failed to heed the advice of Dr. Mallory that morphine, even in small doses, is harmful to patients with cirrhosis of the liver. I should think that his death was incited by the hemorrhage and hastened by the morphine.

I believe that this boy was suffering from cirrhosis of the liver, splenomegaly, and thrombophlebitis of the splenic vein with esophageal varices. The cirrhosis may well have been idiopathic, but the whole picture fits the syndrome described by Banti. The occurrence of varices in syphilis of the liver is unusual. I should hesitate to call this congenital syphilis without additional evidence. It seems that the death might have been premature and that if he had survived the first hemorrhage he might have lived for several years.

Dr. George W. Holmes: The first film shows the characteristic picture of esophageal varices, namely the worm-like shadows of diminished density. In the second film the esophagus looks normal. That frequently happens in cases with

varices, and is a fairly good way to distinguish between deformity of the esophagus due to varices and that due to tumor. In the plain film taken of the abdomen, the edge of the liver is about 2.5 cm. above the crest of the ilium, and there is a large area of increased density in the left upper quadrant, which might be a very much enlarged spleen. There is mottled dullness in the upper part of the right lung field, with similar shadows in the lower part. The bones of the skull, wrists and knees are negative.

A PHYSICIAN: I should like to ask if morphine would be considered adequate cause of the terminal coma. At that age 1/8 gr. might be enough to cause it.

DR. TRACY B. MALLORY: I have been impressed that following a single dose of morphine it is not uncommon for a patient with cirrhosis to develop coma and never come out of it. However, in view of the autopsy findings in this case it would be rash to conclude the morphine was solely responsible.

Dr. Higgins, do you remember Dr. Beth Vincent's findings at the time of operation?

DR. HAROLD L. HIGGINS: The liver felt rough and was not nearly so large as had been thought on clinical examination. Because the patient had jaundice he thought preoperatively that the primary disease was in the liver and expected to find cirrhosis.

CLINICAL DIAGNOSIS

Cirrhosis of the liver ("toxic type" on biopsy).

DR. TALBOTT'S DIAGNOSES

Cirrhosis of the liver, splenomegaly, and thrombophlebitis of the splenic vein.
Banti's syndrome?
Morphine intoxication.

ANATOMICAL DIAGNOSES

Cirrhosis of liver, toxic.
Septicemia, *Streptococcus haemolyticus.*
Fibrosis of spleen.
Otitis media, acute purulent.
Ascites.
Pleural effusion.
Pulmonary congestion, slight.
Acute glomerular nephritis, terminal.
Operative wound: omentopexy.

PATHOLOGICAL DISCUSSION

DR. MALLORY: At the time of autopsy we found a slightly small liver, weighing 800 or 900 gm. It was very nodular and cirrhotic, and the nodules varied from a millimeter to a centimeter in size. On microscopic examination the liver architecture was totally destroyed. The liver cells were grouped in clusters of varying size, without orientation about any central vein to differentiate lobular formation. Between the clusters of liver cells were bands of fibrous tissue containing innumerable bile ducts, which in some areas showed extensive branching, a picture which suggests that multiple contiguous liver lobules had been completely destroyed. In other words, the architecture was essentially that of a healed or nearly healed acute atrophy, with consequent early cirrhosis. There was still some progressive necrosis of the liver cells. The spleen was considerably enlarged, weighing 450 gm., and it showed very early fibrotic changes of the type one finds with any form of portal obstruction. There were no thrombi in the splenic or portal veins.

Microscopic examination provided one surprise. The kidneys showed a very early but definite glomerulonephritis, which I should tie up with a terminal streptococcal sepsis. The blood culture at the time of autopsy showed a beta hemolytic streptococcus.

I should not, personally, regard this case as an example of the currently much debated hepatorenal syndrome. In the few reliably reported necropsies dealing with this disease the lesion has been tubular rather than glomerular.

If I may step out of my role and discuss the differential diagnosis I am sorry that Dr. Talbott did not consider the Banti's syndrome a little more fully. The hematemeses, the esophageal varices, the splenomegaly, the anemia and the persistent leukopenia clearly place this patient in that category, and experience tells us that such patients always have obstruction in the portal system or in the splenic veins. In adults the former is far commoner and is usually the result of cirrhosis of the liver. In children, however, portal and splenic vein thrombosis without cirrhosis occurs with significant frequency and must be given serious consideration. The presence of jaundice was, in this case, strong prima facie evidence in favor of cirrhosis. Had jaundice been lacking, the presence of ascites and the reversed albumin-globulin ratio would have become important. Portal obstruction alone rarely produces ascites unless the serum albumin is lowered, and lowering of the serum albumin is strongly suggestive of hepatic insufficiency.

DR. TALBOTT: There was no evidence of syphilis?

DR. MALLORY: No.

A PHYSICIAN: Have you any explanation for the intermittent character of the jaundice?

DR. MALLORY: No, but I am sure we have seen fluctuations in the level of hepatic incompetence in other cases of cirrhosis.

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ULTRA-VIOLET CURTAINS

The Wellses[1] have conclusively proved that where an aqueous suspension of pathogenic bacteria is thrown into the air by an atomizer the water immediately evaporates and the micro-organisms remain suspended and animate in the air for hours and even days. On the basis of their observations they have advanced the ideas that contaminated air is responsible for the majority of nasopharyngeal infections and that the purification of air by means of ultra-violet radiation is an important factor in the control of such diseases, particularly in hospitals, nurseries and the like; furthermore, a speaker[2] at the last meeting of the American Public Health Association even expressed the belief that influenza and other respiratory diseases could be controlled by means of a curtain of ultra-violet rays. Of these three thoughts, the first appears to be obvious; the second, probable; and the third, fantastic.

Granting that such rays are effective bactericidal and viricidal agents and are excellent for the sterilization of contaminated inanimate objects, such as plates and tumblers, how can they be applied to the infected individual, be he carrier or patient? Again, acknowledging that the bacterium or virus cannot pass through an ultra-violet curtain, how can such curtains be employed to protect a healthy populace? To consider an individual curtain drawn about each carrier or patient is an absurdity. A more pretentious one to protect a nursery full of children or a barrack of soldiers might work if one could be sure that those to be protected never left their haven and that no one from the outside ever entered, both of which are obvious impossibilities. A wall of flame would seem to be just as practical and much more effective.

Attempts along similar lines to prevent sepsis following "clean" operations seem equally futile, for it is a well-known fact that by far the majority of infections following this type of operation are due to micro-organisms from the mouth, nose or throat of the operator or one of his assistants, rather than to those which are circulating in the air of the operating room. The operative field cannot be exposed to ultra-violet rays, because tissue cells also succumb to such radiation. It is conceivable that a horizontal curtain of rays, just above the operating field, might have some effect, but many of the rays would be blocked by hands and instruments.

Until some agent is discovered which is more toxic for bacteria and viruses than it is for other living cells, — including those making up the human body, — epidemics due to diseases of the upper respiratory tract can be controlled only by the strict quarantine of patients, by the elimination of carriers and by measures to increase the specific or non-specific resistance of the populace. Furthermore, it seems likely that unexplained postoperative sepsis can best be prevented by the use of effective masks — if such there be — and by the elimination from the operating team of all those who harbor hemolytic streptococci in their mouths, noses or throats.

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AMERICAN SOCIAL HYGIENE ASSOCIATION

THE far-reaching program of the American Social Hygiene Association is not fully realized until one is confronted by the evidence of its activity. A recent pamphlet issued* by this association shows that taking the population as a whole one person in every twenty is infected with syphilis. This figure is not an exaggeration, and assumes even greater significance if one realizes that the incidence of syphilis is 50 per cent greater than that of tuberculosis, and that there are twenty-eight times as many individuals attacked by syphilis as by infantile paralysis.

The association has performed valiant service in furnishing information about syphilis and gonorrhea to the public. By exhibits, by lectures, by material furnished to newspapers and magazines, by radio, by mail and by moving picture films, wide publicity concerning these diseases has been given to millions of people. The staff members have traveled widely in order to increase contacts with clubs and societies and to set up local social-hygiene groups. The association has been influential in the establishment of premarital and prenatal examinations. Its legal staff has encouraged efficient administration of existing control laws, has made prostitution surveys in many cities and states and has studied medical quackery, particularly with regard to syphilis and gonorrhea, in forty-four cities in twenty-five states. It has furthermore made special studies of conditions near Army and Navy recruiting and training stations.

Its public-health and technical medical consultants have been continuously available to physicians and medical institutions. It has continued its program on sex education and has joined with the United States Public Health Service in the evaluation of health education in schools and colleges.

While encouraged and endorsed by state and federal public-health agencies, all these activities have been supported and made possible by public subscription. Publication of facts and education are half the battle, and it is to be hoped that the funds will be forthcoming to continue this well-organized attack on the problem of genito-infectious diseases. Benefits are accruing, and will still further appear in the form of a decreased incidence of these diseases and fewer serious complications of such infections. This country is indeed fortunate in having an influential, vigorous association conducting such an aggressive campaign against these menaces to public health.

**How Social Hygiene Reached Out to Millions in 1939.* New York: American Social Hygiene Association, 1940.

MEDICAL EPONYM

BORDET-GENGOU BACILLUS

The etiologic agent of whooping cough was described by Jules Bordet and Octave Gengou, of Brussels, in an article entitled "Le microbe de la coqueluche [The microbe of whooping cough]" in the *Annales de l'Institut Pasteur* (**20**: 731-741, 1906). The translation of a portion of the article follows:

> We have been able during the past year to isolate the specific microbe. . . . The authenticity of this microbe as the causal agent of whooping cough depends, to be sure, in large part on the circumstances which existed when it was obtained, but the chief argument seemed to us to be furnished by the study of the specific properties of the serum. The serums of individuals who have not had whooping cough or who had it a considerable time previously, even in large amounts, do not agglutinate the microbe. The serums of children recently recovered from this illness possess an agglutinating power which is of moderate degree, but constant and definite.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

SEPSIS FOLLOWING CESAREAN SECTION

Mrs. P., a thirty-one-year-old primipara at term, started in labor on March 30, 1927. Because of a high fetal head, she was seen in consultation.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

The family history was non-contributory. The past history included diphtheria at the age of three and mastoiditis at fourteen, for which no operation was performed. The tonsils had been removed. There was a history of "heart trouble" after an attack of influenza. Catamenia began at fourteen, were regular with a twenty-eight-day cycle and lasted four days without discomfort. The last period was June 16, 1926, making the expected date of confinement March 23.

The pregnancy had been complicated by Epstein's nephrosis. The urine had contained very large traces of albumin almost all the time, and the blood pressure had ranged from 118 to 130 systolic, 60 to 80 diastolic. She had a large amount of edema and gained 45 pounds in weight. The highest blood pressure recorded was 130 systolic, 60 diastolic, the day before delivery.

Cesarean section was advised. This was done under nitrous oxide, oxygen and ether anesthesia, and an 11 pound, 1 ounce, baby was delivered in good condition.

The day following operation, March 31, the temperature was 99.4°F., with a pulse of 92, and she complained of pain in the right shoulder blade. The temperature rose until it reached 105.6°F. on April 2, with a pulse of 140 and respirations of 48. A medical consultation was held, and an x-ray showed pneumonia in both lungs. The temperature came down to normal on April 6, and vacillated between 98 and 99.6°F. from then until April 15. On April 15 the temperature rose to 101.4°F., and the pulse to 120. At this time there was very definite tenderness throughout the lower abdomen. From April 16 to April 25 the temperature ranged from 99 to 101°F., but it came down to normal on April 25. From April 26 to May 21 the temperature ranged from 98 to 101°F., with a pulse of 90 to 110. During this period the tenderness in the lower abdomen subsided but a definite mass appeared in the epigastrium.

On June 7, because the temperature was still vacillating between 98 and 101°F. and definite induration was felt below the umbilicus on the left, an exploratory laparotomy was undertaken. By this time the mass in the epigastrium had entirely disappeared, and exploration was carried out through an incision to the left of the midline about at the level of the anterior superior spine. No pus was obtained. From the time of operation until June 21 the temperature ranged from 100 to 102°F. From June 22 to June 30 it ranged from 98 to 99.2°F. There was one acute exacerbation on July 6 to 104°F., without anything being found on physical examination. From July 11 until discharge on July 22 the temperature remained normal. During the period from April 15 until ten days before discharge there was a large amount of thin, foul discharge from the vagina; this definitely came from the uterus.

The white-cell counts during this period ranged from 15,000 to 20,000. The urine, save for a large amount of albumin, was always negative. No cultures were taken.

Comment. This case of sepsis following cesarean section began with double pneumonia, a very common complication of Epstein's nephrosis. Whether the infection in the uterus was a blood-stream infection following the pneumonia is debatable. No uterine or blood cultures were taken It is quite likely that at one time or another there was an abscess in the uterine wall which broke into the peritoneal cavity, was walled off and resulted in the definite mass that was felt in the epigastrium. Surgical interference had been considered off and on during convalescence, and was undertaken on June 7, with the idea of draining an abscess cavity and thus shortening the convalescence. This was not achieved. At no time was the uterus invaded.

When seen seven weeks after discharge, the patient's weight had increased from 102 to 123 pounds and she looked very well. The upper two thirds of the cesarean scar presented a hernia. Vaginal examination showed an indefinite fullness on the left, which it was believed would ultimately disappear.

This patient died of nephritis about three years later.

COMMITTEE ON INDUSTRIAL HEALTH

The Committee on Industrial Health has made two reports in which it has brought before the members of the Society what is considered as good practice in industrial medicine. These have been written to assist the general practitioner who is giving part of his time to industrial work.

The committee now proposes from time to time to give short reports of industrial cases. It is hoped that members of the Society will send to the committee similar reports for publication in the *Journal*.

The committee will be glad to answer, to the best of its ability, questions on industrial medicine sent to its secretary: Dr. Louis R. Daniels, Hood Rubber Company, Watertown.

* * *

Moderate Plumbism

F. F., aged 37 years and weight 183 pounds, a battery repairman for 1½ years, reported at the company dispensary on August 10, 1938, complaining of a severe pain in the hypogastric region. He gave a history of some

constipation, moderate loss of weight, weakness and anorexia. Abdominal examination was negative; the pulse and temperature were normal. The outstanding clinical feature was his extreme pallor. There was a suggestive lead line. A tentative diagnosis of plumbism was made. The following day a clinical laboratory reported as follows: "red-cell count 4,500,000; hemoglobin, 80 per cent; smear shows an occasional stippled red cell; red cells are normal in size and shape."

The man was sent home with directions to obtain catharsis with salines, to drink a quart of milk daily and to take belladonna for cramps. In a week he reported that he felt well and that he had had no further abdominal distress. On reporting to work the following day, he was assigned to a job with no lead hazard.

Comment. A case of moderate plumbism is described which occurred in a battery repairman of 1½ years' service, despite the fact that there had been no cases of plumbism for 50 years among the other men similarly employed. His pallor was all out of proportion to his anemia, it was apparent, and the stippled-cell count alone does not establish the diagnosis of plumbism.

For the past year and a half the score of battery repairmen have been examined each month. About 60 per cent of them showed from 2 to 20 stippled cells in 200 oil-immersion fields. None of them have shown any symptoms of plumbism during the period, except one man who showed a lead line daily during a 2-week period when his stippled-cell count jumped from 7 to 32. He was told to drink a quart of milk daily for a couple of weeks while he continued at his job. His stippled count then returned to the original normal level of 1 to 7. Improved housekeeping methods and forced ventilation during the "burning in" of battery plates should prevent further plumbism in the plant.

Conclusions. There is a marked individual susceptibility to plumbism. Extreme pallor with only moderate anemia is indicative of the disease. The mere presence of stippled cells in the blood is not sufficient to make a diagnosis. Workmen exposed to lead who show symptoms of plumbism or a consistently rising stippled-cell curve should be placed where there is no lead hazard.

Dermatitis Venenata

On November 24, 1931, J. F. W., a lineman, reported to the company dispensary, exhibiting a skin rash of the hands which had every appearance of an early poison-ivy dermatitis. He gave a history of picking up old guy wire in the brush along a roadside.

A dermatologist, employed by the insurance carrier, reported the rash to be industrial in origin and probably caused by lack of skin ventilation due to the constant wearing of gauntlet rubber gloves. He described it as follows: "On the backs of both hands and fingers and encircling the wrists is a dermatitis the lesions of which are red, slightly swollen and very itchy."

On February 1, 1932, a similar case appeared at the dispensary, and the same dermatologist noted that the rash appeared after the man had changed from one color of rubber glove to another. When the sixth similar case arrived on March 1, the man brought his gloves with him and the company physician patch-tested himself with a square of the glove; a markedly red, swollen and itching dermatitis developed under the square in about 16 hours. Patch tests done with other makes of gloves at the same time were negative. Therefore, the make of glove which gave the positive patch tests was eliminated, and no further dermatitis has developed.

Comment. This dermatitis was definitely one due to an external irritant, but it took some time to incriminate the gloves, because the make of glove that had caused the dermatitis had been in use for years with no resulting dermatitis. Further investigation revealed that the glove manufacturer had used a new accelerator without previous determination of its irritant qualities. All men who wore the irritant gloves did not develop the rash; however, one man who wore the gloves for only 5 minutes developed a severe rash.

Conclusion. There is a marked variance in individual susceptibility to external irritants which may cause a contact dermatitis.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning April 14:

BERKSHIRE

Thursday, April 18, at 4:30 p.m., at the Bishop House of Mercy Hospital, Pittsfield. Pediatric Institute: Discussion of hospital patients. Instructors: James M. Baty and Elmer W. Barron. Harry G. Mellen, *Chairman.*

BRISTOL SOUTH (Fall River Section)

Tuesday, April 16, at 4:30 p.m., at the Union Hospital, Fall River. Pediatric clinical cases. Instructors: Warren R. Sisson and Lewis W. Hill. Howard P. Sawyer, *Chairman.*

FRANKLIN

Thursday, April 18, at 8:15 p.m., at the Franklin County Hospital, Greenfield. Complications in Obstetrics: Illustrated by case histories. Instructor: Christopher J. Duncan. Halbert G. Stetson, *Chairman.*

HAMPDEN

Thursday, April 18, at 4:00 p.m., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:15 p.m., in the Outpatient Department of the Skinner Clinic, Holyoke Hospital, Holyoke. Common Laboratory Procedures in Pediatrics and Their Interpretation. Instructor: LeRoy D. Fothergill. George L. Schadt, *Chairman.*

HAMPSHIRE

Thursday, April 18, at 4:15 p.m., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Pneumonia. Instructor: Earle M. Chapman. Warren P. Cordes, *Chairman.*

MIDDLESEX SOUTH

Tuesday, April 16, at 4:30 p.m., at the Cambridge Hospital, 330 Mt. Auburn Street, Cambridge. Head and Spine Injuries. Instructor: Walter R. Wegner. Dudley Merrill, *Chairman.*

NORFOLK

Thursday, April 18, at 8:30 p.m., at the Norwood Hospital, Norwood. Head and Spine Injuries. Instructor: Donald Munro. Hugo B. C. Riemer, *Chairman.*

NORFOLK SOUTH

Monday, April 15, at 8:30 p.m., at the Quincy City Hospital, Quincy. Pneumonia. Instructor: Donald S. King. David L. Belding, *Chairman*.

PLYMOUTH

Tuesday, April 16, at 4:00 p.m., in the Nurses' Home of the Brockton Hospital, Brockton. Common Problems of Neurology: Indications for lumbar puncture. Instructor: T. J. C. von Storch. Walter H. Pulsifer, *Chairman*.

SUFFOLK

Thursday, April 18, at 4:30 p.m., in John Ware Hall, Boston Medical Library, 8 Fenway, Boston. Gonorrhea in the Female. Instructor: Alonzo K. Paine. Reginald Fitz, *Chairman*.

ANNUAL PRIZE FOR INTERNS

The attention of interns in Massachusetts hospitals is called to the fact that a prize of $50.00 has been offered by the Massachusetts Medical Society for the best written and most comprehensive case report submitted by one of their number holding an internship in any Massachusetts hospital which is approved by the American Medical Association for intern training during 1938–1940.

This report is to be typewritten, and when completed is to be sealed, unsigned, in a plain envelope, which in turn is to be placed together with a separate slip bearing the name and address of the contestant, in a larger envelope, and sent to: Committee on Medical Education and Medical Diplomas, Massachusetts Medical Society, 8 Fenway, Boston.

The contest this year closes May 5, 1940. Reports may be submitted at any time prior to that date.

DEATHS

DERVIN — Lawrence J. Dervin, M.D., of Somerville, died March 30. He was in his sixty-fifth year.

Born in Clinton, he graduated from Fordham University and received his degree from Ludwig-Maximilians-Universität Medizinische Fakultät in Munich, Germany. Dr. Dervin had practiced medicine in Somerville for thirty-five years. From 1926 to 1930 he was teaching assistant in pediatrics at Tufts College Medical School. Dr. Dervin was a member of the Massachusetts Medical Society, the American Medical Association and the New England Pediatric Society.

His widow, two sons, two daughters and two sisters survive him.

FELCH — Lewis P. Felch, M.D., of Boston, died April 2. He was in his sixty-ninth year.

Born in Nashua, New Hampshire, he graduated from the Massachusetts College of Pharmacy and received his degree from Tufts College Medical School in 1906. He was appointed house officer at the Massachusetts General Hospital in 1909 and was appointed to the staff in 1912, resigning in 1922. He had been medical examiner for the industrial accident board for the past twenty-two years. Dr. Felch was a fellow of the Massachusetts Medical Society and the American Medical Association and held membership in the Boston Orthopedic Club.

His widow, Dr. Carrie Innes Felch, who was in his class at Tufts, survives him.

HILTPOLD — Werner Hiltpold, M.D., of Easthampton, died April 5. He was in his fifty-second year.

Dr. Hiltpold received his degree in 1912 from the University of Vermont College of Medicine. He was a fellow of the Massachusetts Medical Society and the American Medical Association.

MAINS — Herbert L. Mains, M.D., of Danvers, died April 7. He was in his sixty-fourth year.

Born in Salem, New Hampshire, his family moved to Danvers where he attended school. He graduated from Dean Academy and received his degree from the University of Vermont College of Medicine in 1912. He served his internship at Western Hospital in Montreal, Canada, and entered private practice in Danvers. Later he became associated with the United States Veterans' Bureau in Boston and then with various hospitals for tuberculosis and mental patients in New York. He returned to private practice in Danvers in 1936.

Dr. Mains was a member of the Massachusetts Medical Society and the American Medical Association.

His widow survives him.

MORSE — John L. Morse, M.D., of Newton, died April 3. He was in his seventy-sixth year.

Born in Taunton, he attended Harvard University and received his degree from Harvard Medical School in 1891. He served his internship at the Boston City Hospital from 1890 to 1892. Later he served as an assistant in the Department for Diseases of the Nervous System at the Boston Dispensary and did orthopedic work at the Children's Hospital. He was also house physician at the Boston Lying-in Hospital and was registrar of the Carney Hospital and district physician at the Boston Dispensary. For a number of years he served as chief of the medical staffs of the Children's and Infants' hospitals. At the time of his death he was consulting physician at the Children's Hospital, Infants' Hospital, Boston Floating Hospital, Beth Israel Hospital and the North Shore Babies' Hospital.

In 1896 Dr. Morse joined the faculty of the Harvard Medical School as assistant in clinical medicine, and four years later became instructor in clinical medicine. In 1903 he was appointed instructor in pediatrics, in 1906 assistant professor of pediatrics and in 1911 associate professor of pediatrics. In 1915 he became professor and served until 1921, when he became professor emeritus.

Dr. Morse was a fellow of the Massachusetts Medical Society and the American Medical Association. He was twice president of the New England Pediatric Society and the American Pediatric Society and was a co-founder of the American Academy of Pediatrics.

His widow, a son and three grandchildren survive him.

MISCELLANY

MEDICAL STORES BOUGHT BY RED CROSS FOR WAR RELIEF

Medicines, hospital and surgical supplies loom large in American Red Cross war-relief purchases, according to a recent tabulation of articles which that organization is furnishing its sister societies in war-affected countries to assist them in minimizing the sufferings consequent to hostilities.

Analysis of the tabulation, dated April 1, reveals the following purchases: 500,000 tablets of sulfanilamide, 266,000 tablets of sulfapyridine, 100 tons of other assorted drugs, 23,000 surgical instruments, 38 x-ray units, 32 gen-

erating motors for x-ray units, a 100-bed hospital unit, containing 700 items, 25 hospital tents each of 50-patient capacity, 11 motor ambulances, 1,500,000 yards of surgical gauze, 92,000 pounds of absorbent cotton for medical purposes, 276,000 yards of bed sheeting and large quantities of soap, toothbrushes and other similar products.

Purchases for relief also included 120,000 blankets, 105,000 suits of knitted underwear, 45,000 pairs of shoes and various other articles of clothing. In addition, women volunteers in Red Cross chapters all over the country have produced 344,000 garments and 500,000 surgical dressings. These are sent to the Red Cross warehouse in New York, where they are packed and shipped to the various Red Cross societies in the nine European countries currently receiving assistance. These are: Finland, German-occupied Poland, France, England, Latvia, Lithuania, Rumania, Hungary and Yugoslavia. The latter five countries harbor some 122,000 Polish soldiers and civilians who sought refuge there last September.

American Red Cross relief operations in Europe are under the supervision of a commission of two men — James T. Nicholson, from the National Headquarters, Washington, District of Columbia, and Wayne Chatfield-Taylor, former assistant secretary of the treasury. With central offices at Geneva, they are in a position to visit the various relief fronts and personally supervise distribution of supplies and ascertain future needs.

NOTES

Dr. Elliott P. Joslin has recently accepted the honorary chairmanship at Yale University of the Division of Medicine and Public Health of the President's Committee on University Development, succeeding the late Dr. Harvey Cushing. The aim of the committee which Dr. Joslin heads is to secure additional endowment for the medical school so that its facilities may continue to expand, and along with them, its service to the community and the country. The committee also seeks to raise funds for a quadrangle to house medical students.

The appointment of Dr. Charles F. McKhann as professor of pediatrics at the University of Michigan Medical School has been recently announced. Dr. McKhann, who is associate professor of pediatrics at Harvard Medical School, will assume his new duties early this fall.

Dean Stanhope Bayne-Jones, of the Yale University School of Medicine, was elected president of the American Association of Pathologists and Bacteriologists at its recent annual meeting in Pittsburgh. Dr. Bayne-Jones, who succeeds Dr. Carl V. Weller of the University of Michigan, has been vice-president of the organization during the past year and a member of its council since 1937. A member of the executive council of the Association of American Medical Colleges, Dr. Bayne-Jones has been professor of bacteriology at Yale since 1932.

At the recent meeting in Cleveland of the American College of Physicians, Dr. Roger I. Lee, of Boston, was chosen as president-elect. He will assume office, following the expiration of the term of the present president, Dr. James D. Bruce, of Ann Arbor, Michigan, in the spring of 1941.

REPORT OF MEETING

HARVARD MEDICAL SOCIETY

There was a regular meeting of the Harvard Medical Society at the Peter Bent Brigham Hospital on January 9, with Dr. Elliott C. Cutler presiding.

There was one case presentation, by Dr. Harry B. Friedgood. A twenty-nine-year-old single Italian woman first entered the hospital in December, 1938, giving a history of increasing irregularities of menstrual periods since the menarche at the age of fourteen, with a marked change occurring at the age of nineteen. The patient was somewhat obese and hirsute with the onset of the catamenia, but there was a notable increase in both these characteristics ten years before entry. Physical examination on admission revealed a thickset, masculine woman with marked hirsutism of characteristic male distribution, an acneform eruption of the face and back, and an enlarged clitoris. Blood pressure readings varied from 140 systolic, 90 diastolic to 160, 100. X-ray films showed no demonstrable pituitary, adrenal or thymic abnormality. Ten urinary specimens showed an average excretion of 37 mg. equiv. of androsterone in twenty-four hours, as compared with the normal value of 10 to 12 mg. equiv. In view of the finding of a right-lower-quadrant mass considered to be an enlarged ovary and the absence of other localizing signs, a laparotomy was performed by Dr. Robert M. Zollinger. At that time there were found two enlarged ovaries, the right being five times normal size; both were enclosed in thick fibrous capsules. Since the organs were obviously non-functioning clinically, as well as useless by appearance, and since such ovaries are known to produce androgens, a bilateral oophorectomy was performed. Postoperatively the androgen level remained at 34 mg. equiv. and there were no other clinical evidences of improvement, with the possible exception of a slight decrease in blood pressure (130 systolic, 80 diastolic) and a questionable decrease in the growth of hair.

From April to October, 1939, no laboratory data were obtained and the patient's symptoms remained essentially unchanged. At the latter time, however, there was an acute attack of pain considered suggestive of either acute gall-bladder disease or renal colic. The finding of an elevation of the urinary androgen level to over 50 mg. equiv. and the subsistence of symptoms led to further study, which revealed an androgen level persistently above 50 mg. equiv., supposedly in the range of cancerous lesions. Consequently an exploratory operation in the region of the left adrenal gland was performed by Dr. William C. Quinby in December, 1939, and a questionably abnormal organ removed, with the precarious condition of the patient precluding any further exploration. Tests on six specimens of urine since the operation have revealed an average androgen value of 27 mg. equiv., which is approximately half the preoperative level. This was taken as an indication that, at least in this patient, the urinary androgens originated in the adrenal glands and that no tumor was involved. For in the latter instance, the androsterone values would have either fallen promptly to normal in the case of a left-sided tumor or remained essentially unchanged in the event of a right-sided one.

In reply to Dr. Soma Weiss's question as to the validity of assuming that an elevation of the urinary androgen levels above 50 mg. equiv. indicates a malignant neoplasm, Dr. Friedgood stated that the chemical identity of the excreted androgens was probably a more significant finding. In cancer the percentage of dehydroisoandrosterone approaches 50 per cent of the total urinary androgen output, whereas the usual value is only 5 per cent.

The pathological department reported that the ovaries were very large, with dense fibrous-tissue capsules and no evidence of ovulation. The removed adrenal gland was within normal weight when due account was taken

of a large medullary hemorrhage. There was no evidence of neoplasia in any organ.

The speaker of the evening was Dr. H. Stanley Bennett, whose subject was "The Mechanism of Secretion in the Adrenal Gland." It was pointed out by way of introduction that the adrenal gland in mammals is really composed of two separate organs, each with its own secretion. Although the possibility of there being some chemical interrelation between these parts of the gland has been postulated, the presence of the cortical hormones from isolated nodules of cortex and the probable presence of adrenaline in the abdominal paraganglia obviate the necessity of any such hypothesis, and indeed make it untenable. Furthermore, fish, reptiles and amphibians have no such close spatial relation to these two organs.

Since it has been fairly conclusively demonstrated that the adrenocortical sterones are all derivates of cholesterol and can maintain adrenalectomized animals, and since it has been shown that the ketone fraction of the adrenal gland contains all this biologic activity, it was attempted to determine by color reactions the localization of the biologically active ketones in the adrenal cortex. In order to further clarify the mechanism of secretion, the blood vessel configuration in the gland was presented, for it was concluded that the hormone must exit via this channel since the lymphatics run only to the capsule. The audience was also reminded that the three zones of the adrenal cortex are in reality three phases of the life cycle of the cortical cells, which are formed in the subcapsular region and migrate inward to their senescence in the juxtamedullary portion of the gland.

By employing sudan III and osmic acid, Dr. Bennett was able to differentiate four rather than three zones in the adrenal cortex of the cat. The application of phenylhydrazine or ammoniacal silver solution to frozen sections showed that water-insoluble ketones were confined to the second of these four zones — the outer portion of the fasciculata — and that the biologically active sterones are hence localized in this area, which could be regarded as the secretory zone of the cortex. The specific ketone nature and the solubility properties of the compounds giving rise to the phenylhydrazone reaction were demonstrated by the prevention of the formation of the yellow color of the phenylhydrazones following previous acetone or alcohol extraction or by causing the sections to be immersed in a semicarbazide solution. Further observation of sections in which the cholesterol was precipitated by digitonin showed that the above-mentioned secretory zone is also richest in cholesterol and its esters. This is consistent with Fieser's assertion that cholesterol is a precursor of the steroid hormones. The four zones revealed in the cat's adrenal cortex by these methods were designated as presecretory, secretory, postsecretory and senescent, in accordance with the supposed functional state of the cells comprising the respective zones.

Dr. Bennett exhibited drawings demonstrating the cytology of the various zones, particularly the great increase in cell size and the number of secretory lipoid droplets during the secretory phase. The findings were found consistently in all mammals when due consideration was made for slight quantitative variations of species.

A study of the blood supply revealed a rich radial plexiform arrangement of capillaries streaming from the capsule toward the medulla through the cortex.

Dr. Bennett inaugurated the discussion of the medulla by reminding his audience of the chromaffin reaction, whereby a number of oxidizing agents will cause that part of the gland which contains adrenaline to be stained brown. This was considered to be due to the oxidation of the adrenaline to an insoluble brown substance, and the intensity of the brown reaction was thought to vary in proportion to the amount of adrenaline in the cells, thus accounting for the patchy character of the staining reaction. Ammoniacal silver solution was used to accentuate the varying color intensity and was of particular advantage in photographing sections of the gland.

A review of the medullary blood supply revealed that it arises from two sources: the cortical capillaries, which drain into an extensive branching venous tree in the medulla, and the direct radial medullary arteries, which pass from the capsule through the cortex to the medulla and break up into a fine capillary bed between the venous radicals in the medulla. A close analysis of the relation of the cells to the vascular system revealed the important fact that there was characteristically a true polarity of cells, with one end of the cell directed toward the arterially supplied capillaries and the other toward the veins. Properly prepared sections showed that the medullary cells were typically arranged as columnar epithelium along the veins, and that this arrangement gives rise to the appearance of the conventionally described cords and whorls in glands fixed by immersion. Further investigation disclosed that the nucleus was always nearer the capillary pole of the cell, while the Golgi apparatus and secretion droplets occupied a position between the nucleus and the venous pole. It was concluded, therefore, that the venous pole was predominantly secretory, whereas the opposite capillary pole was primarily nutrient. Cytological changes in the cells were correlated with different secretory phases of the cells, and the hypothesis was advanced that the medullary cells go through a series of cycles of secretion, in the course of which one can identify phases of storage of adrenaline, secretion, exhaustion and regeneration.

NOTICES

REMOVALS

ISRAEL KOPP, M.D., announces the removal of his office to 353 Commonwealth Avenue, Boston.

THEODORE J. C. VON STORCH, M.D., announces the removal of his office to the Robert Dawson Evans Memorial, Department of Clinical Research and Preventive Medicine, of the Massachusetts Memorial Hospitals, 78 East Concord Street, Boston.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, April 23, in the amphitheater of the Peter Bent Brigham Hospital (Shattuck Street entrance), at 8:15 p.m. Dr. Elliott C. Cutler will preside.

PROGRAM

Some Recent Advances in Our Knowledge of the Biliary Tract. Dr. I. S. Ravdin, of Philadelphia.

Medical students and physicians are cordially invited to attend.

BOSTON MEDICAL HISTORY CLUB

There will be a meeting of the Boston Medical History Club at the Boston Medical Library, 8 Fenway, Boston, on Monday evening, April 15, at 8:15. Dr. Robert B. Osgood will speak on "Menders of the Maimed."

All those interested in the subject are cordially invited to attend.

PETER BENT BRIGHAM HOSPITAL

A joint medical and surgical clinic at the Peter Bent Brigham Hospital will be held on Wednesday, April 17, from 2 to 4 p.m. Drs. Elliott C. Cutler and John Romano will speak on "Headache."

Physicians and students are cordially invited to attend.

HARVARD MEDICAL SCHOOL LECTURE

The annual lecture on "The Care of the Patient" will be given by Dr. Charles R. Austrian, of Johns Hopkins University School of Medicine, at the Harvard Medical School, Amphitheater C, on Monday, April 15, at 5 p.m.

COMMUNITY NURSING COUNCIL OF BOSTON

The second annual meeting and luncheon of the Community Nursing Council of Boston will be held at the Young Women's Christian Association, 140 Clarendon Street, Boston, on Tuesday, April 23, at 12:30. Dr. Channing Frothingham will speak on "An Experiment in the Delivery of Medical Care."

The charge for luncheon will be 85 cents. Reservations, sent to the Community Nursing Council of Boston, 80 Federal Street, Boston, should be made by Saturday, April 20.

QUINCY CITY HOSPITAL

The fiftieth anniversary of the founding of the Quincy City Hospital will be celebrated on April 18 and 19. The program is as follows:

THURSDAY, APRIL 18

9:00–10:30 (Administration Building). Symposium on Fractures. Dr. A. W. Reggio and associates.

10:40–12:00 (Administration Building). Symposium on Obstetrics. Dr. F. L. Good and associates.

12:10–1:25 (Administration Building). Appendicitis Cases at the Quincy City Hospital. Dr. J. E. Knowlton.

1:30 (Nurses' Dining Room). Steak dinner for the members of the medical staff and of the Norfolk South District Medical Society.

4:00 (Administration Building). Blood Dyscrasias. Dr. W. P. Murphy.

FRIDAY, APRIL 19

9:00–10:30 (Conference Room, Administration Building). Panel Discussion: Modern surgery of the gall bladder. Conducted by Dr. W. R. Hurley.

12:15 (Neighborhood Club). Luncheon, to be followed by:

The Place of the Community Hospital in Any Public-Health Program. Dr. A. S. Pope.

History of Medicine. Tableaux presented by the graduates and students of the School of Nursing.

1940 Trends in Nursing. Elizabeth Sullivan, R.N.

7:00 (Neighborhood Club). Formal dinner. Dr. F. R. Burke, toastmaster; invocation by the Rt. Rev. Michael J. Owens, V.F. The scheduled speakers and their subjects are as follows:

History of the Hospital. Dr. C. J. Lynch.

Highlights in the Development of the Hospital. Drs. Burke, Reardon, McCausland, Hurley, Sargent, Adams and Lynch.

Present-Day Résumé of the Hospital. Mr. G. W. Hart, chairman of the Board of Managers.

The Role of Hospitals in the Progress of Surgery in the Last Half Century. Dr. E. C. Cutler.

Anticipated Future Developments. Dr. J. P. Leone.

NEW ENGLAND SOCIETY OF PSYCHIATRY

The annual meeting of the New England Society of Psychiatry will be held at Danvers State Hospital, Hathorne, Massachusetts, on Thursday, April 25.

PROGRAM

10:00 a.m.–1:00 p.m. Inspection of hospital.

1:00 p.m. Luncheon and business meeting. Dr. M. Ralph Kaufman will speak on "Factors in Psychotherapy: A psychoanalytic evaluation."

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

Because of the coming scientific session of the Academy of Physical Medicine at Richmond, Virginia, April 24 to 26, there will be no regular scientific program of the New England Society of Physical Medicine this month. Members will meet for a business session at the Hotel Kenmore on April 17.

NEW ENGLAND PATHOLOGICAL SOCIETY

The next meeting of the New England Pathological Society will be held at the Peter Bent Brigham Hospital on Thursday, April 18, at 8:00 p.m. Dr. Hamilton Montgomery, a member of the Section on Dermatology, Mayo Clinic, will speak on the subject "Pathologic Features of Some of the Non-Neoplastic Dermatoses."

Physicians and medical students are cordially invited.

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS

The twenty-fifth annual meeting of the American Association of Industrial Physicians and Surgeons, together with the first annual meeting of the American Industrial Hygiene Association, will be held at Hotel Pennsylvania, New York City, June 4, 5, 6 and 7. This will be a four-day convention intensively devoted to the problems of industrial health in all their various medical, technical and hygienic phases, with particular stress on the prevention and control of occupational hazards. Important programs have been prepared, and technical and scientific exhibits will be a feature. The dinner on Thursday evening, June 6, will be the occasion of the presentation of the William S. Knudsen award for the year 1939–40. The medical profession is not only invited but urged to attend these gatherings as they will be of unusual interest and value to all practitioners interested in industrial injuries and illnesses.

ACADEMY OF PHYSICAL MEDICINE

The eighteenth annual meeting and scientific session of the Academy of Physical Medicine will be held at Richmond, Virginia, April 24, 25 and 26, under the presidency of Dr. Harold D. Corbusier, of Plainfield, New Jersey. The headquarters will be at the Hotel John Marshall, with demonstrations and clinics at the Academy of Medicine Building. Brigadier General Frank T. Hines, chief of the United States Veterans' Administration, will be the speaker at the annual banquet on the evening of April 25. Dr. J. Fulmer Bright, Mayor of Richmond, and other men of prominence will address the public meeting. There will be extensive exhibits. An attractive social program has

been planned for the ladies under the direction of Mrs. Thomas Wheeldon. The session occurs during Garden Week in Richmond.

The scientific program will be open to the medical profession without registration fee.

The program will be forwarded on request, addressed to Herman A. Osgood, M.D., Secretary, 144 Commonwealth Avenue, Boston.

AMERICAN PUBLIC HEALTH ASSOCIATION

The sixty-ninth annual meeting of the American Public Health Association will be held in Detroit, Michigan, October 8–11, with the Book-Cadillac Hotel as headquarters. The Michigan Public Health Association, the American School Health Association, the International Society of Medical Health Officers, the Association of Women in Public Health and a number of other allied and related organizations will meet in conjunction with the association. The Michigan Committee on Arrangements is headed by Mr. Abner Larned, of Detroit. Dr. Henry F. Vaughan, health commissioner of Detroit, is executive secretary.

The annual meeting of the American Public Health Association is the largest and most important health convention held on this continent. It will bring 3500 health officials to Detroit for a series of scientific meetings covering all phases of health protection and promotion. A Health Exhibit will be held in connection with the meeting, and an Institute on Health Education is scheduled prior to the official opening.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Sunday, April 14

Monday, April 15

5 p.m. The Care of the Patient. Dr. Charles R. Austrian. Harvard Medical School, Amphitheater C.

*8:15 p.m. Menders of the Maimed. Dr. Robert B. Osgood. Boston Medical History Club. Boston Medical Library, 8 Fenway.

Tuesday, April 16

*9–10 a.m. The Origin, Diagnosis and Treatment of Pituitary Body Tumors. Dr. Oscar Hirsch. Joseph H. Pratt Diagnostic Hospital.

*12 m. Surgery of the Lungs. Dr. Edward D. Churchill. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

*8:15 p.m. The Heredity of Mental and Nervous Disease and Eugenical Sterilization. Dr. Abraham Myerson. Boston Lying-in Hospital, Journal Club meeting.

Wednesday, April 17

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*2–4 p.m. Headache. Drs. Elliott C. Cutler and John Romano. Peter Bent Brigham Hospital.

Thursday, April 18

*9–10 a.m. Gastrointestinal Clinic. Drs. K. S. Andrews, H. H. Lerner and L. M. Asher. Joseph H. Pratt Diagnostic Hospital.

*8 p.m. Pathologic Features of Some of the Non-Neoplastic Dermatoses. Dr. Hamilton Montgomery. New England Pathological Society. Peter Bent Brigham Hospital.

Saturday, April 20

*9–10 a.m. Hospital case presentation. Dr. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

April 15–17 — American Association for the Study of Goiter. Page 203, issue of February 1.

April 15–19 — New England Health Institute. Page 284, issue of February 15, and page 608, issue of April 4.

April 18 and 19 — Quincy City Hospital. Page 654.

April 23 — Harvard Medical Society. Page 653.

April 23 — Community Nursing Council of Boston. Page 654.

April 24 — Massachusetts Dental Society. Page 365, issue of February 29.

April 24–26 — Scientific Session. Academy of Physical Medicine. Page 654.

April 25 — New England Society of Psychiatry. Page 654.

April 26 — Sir William Osler Honorary Society of the Tufts College Medical School. Page 607, issue of April 4.

May 5 — Boston Doctors' Symphony Orchestra concert. Page 607, issue of April 4.

May 9 — Pentucket Association of Physicians. 8:30 p.m., Hotel Bartlett, Haverhill.

May 10–18 — American Scientific Congress. Page 1043, issue of December 28.

May 13 — United States Pharmacopoeial Convention. Page 202, issue of February 1.

June 4–7 — American Association of Industrial Physicians and Surgeons. Page 654.

June 7–8 — American Heart Association. Page 469, issue of March 14.

June 7–10 — American Board of Obstetrics and Gynecology. Page 608.

June 8 and 10 — American Board of Ophthalmology. Page 719, issue of November 2.

June 10–14 — American Physicians' Art Association. Page 332, issue of February 22.

June 23–25 — Maine Medical Association. Annual meeting. Rangeley Lakes.

October 8–11 — American Public Health Association. Notice above.

October 21 — American Board of Internal Medicine, Inc. Page 369, issue of February 29.

District Medical Societies

ESSEX SOUTH

May 8 — Annual meeting. Salem Country Club, Peabody.

FRANKLIN

May 14 — Franklin County Hospital, Greenfield.

HAMPSHIRE

May 8, at 11:30 a.m. at the Cooley Dickinson Hospital, Northampton.

MIDDLESEX EAST

May 15, at 12:15 p.m. at the Unicorn Country Club, Stoneham.

MIDDLESEX NORTH

April 24.
July 31.
October 30.

NORFOLK SOUTH

May 2.

PLYMOUTH

April 18 — State Farm.
May 16 — Lakeville State Sanatorium, Middleboro.

SUFFOLK

April 24 — Annual meeting in conjunction with the Boston Medical Library. Election of officers. Program and speakers to be announced later.
May 2 — Censors' meeting. Page 244, issue of February 8.

WORCESTER

May 8 — Worcester Country Club. Dinner at 6:30 p.m. followed by a business and scientific meeting.

BOOKS RECEIVED FOR REVIEW

Fetal and Neonatal Death. Edith L. Potter and Fred L. Adair. 207 pp. Chicago: University of Chicago Press, 1940. $1.50.

The New International Clinics: Original contributions, clinics and evaluated reviews of current advances in the medical arts. Edited by George M. Piersol. Vol. 1, N. S. 3. 319 pp. Philadelphia, Montreal and New York: J. B. Lippincott Co., 1940. $3.00.

The Newer Nutrition in Pediatric Practice. I. Newton Kugelmass. 1155 pp. Philadelphia, Montreal and London: J. B. Lippincott Co., 1940. $10.00.

Traité de l'immunité dans les maladies infectieuses. Jules Bordet. Second edition. 879 pp. Paris: Masson et Cie, 1939. $3.90.

Elmer and Rose: Physical diagnosis. Revised by Harry Walker. Eighth edition. 792 pp. St. Louis: C. V. Mosby Co., 1940. $8.75.

Obstetrics and Gynecology: By the departmental staff of the University of Chicago and other contributors. Edited by Fred L. Adair. 2 vol. 2031 pp. Philadelphia: Lea & Febiger, 1940. $20.00.

Transactions of the American Gynecological Society—1939. Vol. 64. Edited by Richard W. TeLinde. 298 pp. St. Louis: C. V. Mosby Co., 1940.

Clinical Roentgenology of the Alimentary Tract. Jacob Buckstein. 652 pp. Philadelphia and London: W. B. Saunders Co., 1940. $10.00.

Essentials of the Diagnostic Examination. John B. Youmans. 417 pp. New York: The Commonwealth Fund, 1940. $3.00.

Dermatologic Allergy: An introduction in the form of a series of lectures. Marion B. Sulzberger. 540 pp. Springfield, Illinois, and Baltimore: Charles C Thomas, 1940. $8.50.

The Pathology of Internal Diseases. William Boyd. Third edition. 874 pp. Philadelphia: Lea & Febiger, 1940. $10.00.

BOOK REVIEWS

Injuries of the Nervous System: Including poisonings. Otto Marburg and Max Helfand. 213 pp. New York: Veritas Press, 1939. $3.00.

The senior author was for many years head of the Neurologisches Institut der Universität, in Vienna, and more recently has been connected with the Montefiore Hospital in New York. His work on the pathology of the nervous system is well known, and he has been one of the outstanding contributors to this subject in the last few decades. A book with such a distinguished man as one of the authors, therefore, needs careful perusal. The subject is carefully investigated with a reference list covering most of the literature published in recent years. One misses, however, one of the most important books on intracranial injuries issued in America in the last few years, namely that by Donald Munro.

The fields of symptomatology, pathology and treatment are considered both for cerebral injuries and for those of peripheral nerves. In addition, there are chapters on electric injuries, caisson disease, trauma as a cause of organic nervous diseases and poisonings. In a book of slightly over two hundred pages, the reviewer believes that the authors have tried to cover too much ground. Much of their material is presented in a sketchy manner, particularly that of the clinical manifestations of injuries and the sections on treatment. The part dealing with pathology is excellent, and there are a number of important illustrations. An interesting chapter deals with the relations between trauma and organic disease. As with most neurologists, the authors take the middle ground, pointing out that trauma may be a factor in almost any structural disease of the nervous system, although the incidence is small.

In general, the book reviews the subjects in a fairly adequate manner. Much of it is written, however, in the on-the-other-hand style; a definite stand is not taken, a position which would have been welcome in view of the well-earned reputation of the senior author. The book is well printed, and the illustrations are adequate. There is a moderately extensive bibliography and index. It is a book which no neurologist should fail to read. It makes an interesting preparation for medicolegal testimony. It is not quite extensive enough, however, to be quoted and one hopes that the authors will write a book in the near future covering the subject in detail. There are a few minor errors in spelling, such as, for instance, the name of Dr. Naffziger.

Diagnostic Signs, Reflexes and Syndromes. W. Egbert Robertson and Harold F. Robertson. 309 pp. Philadelphia: F. A. Davis Co., 1939. $3.50.

This book is a valuable reference work. No one has heretofore undertaken to accumulate in a single volume the various signs, reflexes and syndromes, predominantly eponymic. In this volume they are arranged alphabetically and accompanied by a brief definition.

The appearance of this book is a significant event not because it is a definitive work but rather because it raises an issue which has been a source of controversy in the medical profession. The history of science is replete with manifestations of veneration for its pioneers. Nowhere is it carried to the degree found in the field of medicine. As is evidenced by this book we now find ourselves with a heritage of hundreds of non-descriptive eponyms impinging on the minds of the profession, obfuscating students and burdening medical literature. On the one hand is the sincere realization that the classical descriptions of the original observers warrant historical notation and recognition; on the other hand is the crying need for reorientation and simplification of medical terminology. The latter is illustrated in the following quotation from page 43 of the June, 1934, volume of *International Clinics* (Philadelphia, Montreal, London: J. B. Lippincott Company):

> Nonhemophilic, nonpurpuric, and nonthrombopathic heredofamilial bleeding with or without (telangiectasia) heredofamilial angiomatosis (Rendu-Osler-Weber's Disease or Goldstein's Disease), hemophilia, thrombasthenic and thrombopenic purpura, Frank's pseudohemophilia hepatica ("hypoleukia splenicohepatica") and hemorrhagic capillary toxicosis, Von Willebrand's hepatogenic hemorrhagic diathesis, Jürgen and Von Willebrand's constitutional thrombopathy, David's hemorrhagic (dysendocrinism) disease in women, metropathia haemorrhagica, Glanzmann's familial thrombasthenic purpura, pseudohemophilia, and Biermer's hyperchromic macrocytic anemia, severe hypochromic microcytic anemia have, during recent years, received considerable attention.

Until some simplified form of medical terminology is adopted, it would be well to possess this volume, which is published in a handy size and is thumb indexed.

Civilization against Cancer. Clarence C. Little. 150 pp. New York and Toronto: Farrar & Rinehart, Inc., 1939. $1.50.

Any author will realize the obvious difficulties expected in writing a short book on cancer for the lay public. The inescapable limits to this subject, theoretical and practical, are so involved in details from all branches of modern science that it requires considerable skill to present the material in concise and understandable language. It must be admitted that the author has accomplished this singular feat, leaving out enough of the detail and selecting facts to support the educational program of cancer control. The book is interesting, contains a minimal amount of personal theories and reflects in a modest way the author's abilities in the field of organized cancer research. It is to be recommended to all and sundry and should prove of value in the dissemination of knowledge in this branch of medicine. The book when read will not frighten anyone, or subject him to any of the unusual fears of neoplastic disease.